

GASTRO-INTESTINAL OBSTRUCTION

By MEYER O CANTOR, MD MS FACS

*Associate Attending Surgeon Grace Hospital Attending Surgeon Sinai
Hospital of Detroit Former Attending Surgeon Deaconess Hospital*

and ROLAND P REYNOLDS MD FACS

Chief of Surgery Grace Hospital

Contributors

CLIFFORD D BENSON MD FACS

*Associate Professor of Surgery Wayne University Vice chief Children's Hospital of
Michigan Attending Surgeon Harper Hospital*

ROBERT E L BERRY MD FACS

Associate Professor of Surgery University of Michigan

WILLIAM A HUDSON MD MS FACS

*Associate Professor Surgery (retired) Wayne University Chief of Staff Metropolitan
Hospital and Clinics Past President American College of Chest Physicians*



THE WILLIAMS & WILKINS COMPANY
BALTIMORE 1957

Copyright © 1947
THE WILLIAMS & WILKINS COMPANY
Made in the United States of America

Library of Congress
Catalog Card Number
57 10292

*Dedicated to Our
Sons, Robert*

The improvement in the mortality rate for intestinal obstruction is considered to be due largely to the use of intestinal decompression the judicious use of blood and antibiotics and our understanding of water and electrolyte balance. However the present mortality rate of 10 to 15 per cent is still quite appreciable and the improvement attained by the use of these newer agents will have little effect in further lowering this rate. Any further reduction in mortality for this dread disease must be brought about by a better understanding of intestinal obstruction and by earlier diagnosis. The personal equation and the ability of the attending physician to diagnose and treat intestinal obstruction earlier is our only hope for further improvement.

No surgeon not even one of vast experience can always be certain as to whether or not a strangulating obstruction is present. Nor in every case can one state with certainty at what moment an operation may be required. Even if one's judgement has been correct in many instances the very next patient may demonstrate the fallibility of this judgement.

The acute type of intestinal obstruction presents one of the most urgent diagnostic problems. The element of time is a critical factor. The mortality increases with each hour of delay. Many early writers on this subject have stated that the mortality rate increases 1 per cent for each hour that the obstruction exists. It is universally agreed that those patients who are treated early in the course of the disease may all be expected to recover. In fact it might be said that brilliant results can be obtained if patients with intestinal obstruction receive surgical attention before strangulation or peritonitis occurs. For those patients in whom the diagnosis is made late the intestinal obstruction *per se* is merely one of a series of conditions requiring treatment. Because of this it should be apparent that early diagnosis and early treatment are the *sine qua non* to good results in the management of intestinal obstruction.

Our purpose in producing this book on gastro

intestinal obstruction is to coordinate the various branches of medicine such as radiology, internal medicine and surgery each of which plays a very important role in the successful treatment of advanced cases of intestinal obstruction.

After many years of practice and observation we believe that although the laboratory must be used to aid the clinical diagnosis it should not supersede it. The clinical examination of the patient and a carefully taken history are of the greatest importance in diagnosing this disease.

Since the general practitioner is often the first man to see the patient the burden of early diagnosis frequently rests upon him. For this reason we are attempting to bring together in a rather concise form all the available knowledge on the subject so that it may be useful to the general practitioner and the specialist alike. The onset of acute intestinal obstruction may be so treacherous that even the surgeon suspecting the possibility of its presence may fail to detect it in time for early treatment. Although it has been repeatedly emphasized that intestinal obstruction is the one disease which should be operated upon even on suspicion it is interesting to note in some reports that those cases of obstruction which developed while under observation in the wards of the hospital had a mortality rate 10 per cent higher than the general rate for this disease. This emphasizes the fact that it is not the fault of the family physician alone and that cooperation between the patient the internist the surgeon and the radiologist is necessary if the best results are to be obtained.

While it is not at all unusual for a patient admitted to a ward to be studied carefully intubated slowly and watched under a sit tight policy until an accurate and complete diagnosis is made it has been demonstrated rather conclusively that this policy kills as many patients with intestinal obstruction as does the use of morphine and cathartics.

We are firmly convinced that all cases of intestinal obstruction of a mechanical nature should

be operated upon as soon as they can be properly prepared. This may require as little as 6 hours or as long as 24 hours after admission. During this period of time dehydration, electrolyte imbalance, and restoration of blood volume should be corrected to as near normal as possible. Intestinal distention may be considerably improved by the proper use of the long intestinal decompression tube. In those cases in which a delay in the downward passage of the tube exists, the intestinal de-

compression sound and the liberal use of antibiotics make it possible to decompress the patient at the time of surgery with a minimum of risk and a maximum of benefit.

The nature of this work is such that a certain amount of repetition is necessary to insure continuity of thought. Moreover, we believe that important points can be emphasized by this method. However, we have attempted to avoid excessive repetition.

ACKNOWLEDGMENTS

We but stand on the shoulders of those who went before us

In the course of compiling this work for which thousands of articles were read and the literature reviewed it was found that approximately 18,000 articles on intestinal obstruction had accumulated in the literature. Many thousands of surgeons and physicians contributed to this gigantic amount of literature. We would be remiss if we did not acknowledge our debt to all of these men who went before us and contributed to our understanding of this subject.

In addition to the great host of men whose writings were reviewed we are especially indebted to the following, for permission to use material and for furnishing illustrations: I. W. Burd, J. P. Barnes, J. Larron, J. M. Beardsley, D. Braxton, J. A. Buckwalter, R. P. Cattell, J. S. Chaffee, J. W. Chamberlain, I. B. Cockett, O. H. Cohen, B. I. Colecock, D. C. Collins, J. F. Connolly, T. S. Cullen, J. W. Devine, J. W. Devine, Jr., C. K. Edwards, I. R. Fairchild, L. Figiel, S. Engel, C. I. Gardner, Jr., J. H. Clifford, B. A. Glass, W. Gissane, H. K. Gray, M. T. Green, I. J. Gregory, R. F. Gross, K. B. Hatt, J. W. Hinton, S. O. Hoerr, C. J. Hunt, B. R. Kirklin, I. J. Kleinsasser, I. C. Krieg, M. I. Lichtenstein, I. A. Manelli, A. M. May, H. W. Mayo, Jr., W. M. McMillan, J. B. Millet, C. G. Morlock, J. J. Morton, M. Musselmann, J. K. Nivat, R. M. Tool, R. J. Iriest, W. I. Quinn, J. Rabinovitch, S. Rabinovitch, J. T. Rose, I. R. Schwartz, F. A. R. Stammers, H. C. Stephenson, Jr., W. C. Sullivan, P. Thorek, E. Torre, F. C. Usher, O. H. Wangenstein, K. W. Warren, L. F. Watson, J. P. West, S. D. Weston, P. D. Wilson, R. L. Zech.

We are grateful to The Charles C. Thomas Company, The Lippincott Company, The Saunders Company, The Harvard University Press, and Lea Febiger Company for permitting us to

use illustrations previously published by them and to which they hold a copyright.

We also wish to thank the following journals for permission to use illustrations previously printed in them: The American Journal of Surgery, American Journal of Radiology and Roentgen Therapy, American Journal of Digestive Diseases, Archives of Surgery, The British Journal of Medicine, The British Journal of Surgery, Journal of the American Medical Association, Radiology, and Gastroenterology. Thanks are due the Dohy Chemical Corporation for the use of illustrations from Weston's *Proctologies*.

We are indebted to Dr. H. Jarre, chief of radiology, Grace Hospital, for most of the radiologic illustrations.

Kosa Lynn Epstein was especially helpful in putting together the accumulated material.

Above all, we wish to express our appreciation to The Williams & Wilkins Company for being so understanding and helpful during the final stages of this work. We especially wish to thank Mr. Dick M. Hoover for his courtesy and unfailing optimism. We are also deeply indebted to Mary MacIsaac for her kindness and understanding assistance at the crucial point in revising the manuscript. This made it possible for the authors to complete the work in a relaxed frame of mind. Finally, without the able help of Mary Whitney Allen of the editorial department, this book as it stands would not have been possible. Her great understanding of the subject and the application of a brilliant ability is responsible for any success that this book may have.

We sincerely hope that we have acknowledged our debt to all those whose material was useful in the compilation of this book. In the event that anyone has been inadvertently omitted, we wish to apologize for the unintentional oversight.

M O C
R P R

Table of Contents

1 HISTORY OF GASTRO INTESTINAL OBSTRUCTION	1
Antiquity The 16th century The 17th century The 18th century The 19th century The 20th century	
2 EMBRYOLOGY OF THE GASTRO INTESTINAL TRACT	7
Esophagus Stomach Duodenum and pancreas Small intestine and colon Anus and rectum Intestinal lumen Myenteric plexuses and ganglia Mesenteries Bursae	
3 ANATOMY OF THE GASTRO INTESTINAL TRACT	18
Esophagus Esophagus Stomach Duodenum Ligament of Treitz Duodenal fossae Small intestine Ileocecal valve Large intestine Rectum and anus Ileocecal and sigmoid fossae Veils Bands and adhesions Peritoneal fat line	
4 PHYSIOLOGY OF THE GASTRO INTESTINAL TRACT	36
Mechanism of deglutition Stomach reservoir and sphincters Duodenum Small intestine Ileocecal valve Colon Transit time from mouth to anus	
5 OBSTRUCTION OF THE ESOPHAGUS	46
Symptoms Diagnostic procedures Obstructive diseases and their treatment	
6 OBSTRUCTION OF THE STOMACH AND SPHINCTERS	61
Cardiospasm Stenosis at the cardia Infections Foreign bodies Congenital anomalies Neurogenic causes Neoplastic causes Gastric piles Ulcers Postoperative causes Volvulus Intussusception Obstruction associated with diaphragmatic hernia Hypertrophic pyloric stenosis in the adult	
7 OBSTRUCTION OF THE DUODENUM	74
Adhesions Arterioesenteric compression Anomalies Extramural compression Foreign bodies Diverticula Infections Neoplasms Neuromuscular disorders Ulceration	
8 OBSTRUCTION OF THE SMALL INTESTINE	88
Diagnosis Signs and symptoms Nonstrangulating versus strangulating obstruction Etiology	
9 OBSTRUCTION OF THE COLON	157
Diagnosis Role of the ileocecal valve Etiology	
10 OBSTRUCTION OF THE RECTUM AND ANUS	197
Infections Foreign bodies Postoperative causes Congenital anomalies Pressure of pelvic tumors or masses Neoplasms Fecal impaction X-ray irradiation Metastatic growths Sigmoidorectal intussusception	
11 CHRONIC INTESTINAL OBSTRUCTION	204
Chronic obstruction of the stomach Chronic duodenal obstruction Chronic small bowel obstruction Chronic obstruction of the colon	
12 INTESTINAL OBSTRUCTION COMPLICATING OTHER MAJOR PATHOLOGY	210
13 OBSTRUCTION IN INFANCY AND CHILDHOOD	214
Technic of examination General management of intestinal obstruction Etiology and management of obstruction in infancy and childhood	
14 GASTRO INTESTINAL OBSTRUCTION AND PREGNANCY	242
Diagnosis Causes of obstruction Management of obstruction in pregnancy	
15 OBSTRUCTION IN THE AGED	250
Associated problems in the management of the elderly obstructed patient Examination and diagnosis Factors and risks accompanying surgery Etiology and treatment Postoperative treatment	
16 RADIOLOGIC DIAGNOSIS OF GASTRO INTESTINAL OBSTRUCTION	261
Radiologic diagnosis via gas patterns Radiologic diagnosis by other media	

17	DISTENTION IN THE GASTRO-INTESTINAL TRACT	293
	Sources of gas causing distention Composition of gas within the bowel Composition of intestinal content (other than gas) causing distention Cause of intestinal distention Effects of intestinal distention Effect of intestinal gases upon balloons of intestinal decompression tubes	
18	GASTRO-INTESTINAL INTUBATION	314
	Use of gastroduodenal tubes Use of the long intestinal decompression tube Suction devices and their use in bowel obstruction Radiologic intestinal intubation Surgical intubation The use of intestinal decompression sound Length of intestinal decompression tube required Indications for use of intestinal decompression tube Contra-indications to the use of the long intestinal decompression tube Complications due to the use of the long intestinal decompression tube The use of mercury in the treatment of intestinal obstruction Balloons of long intestinal decompression tubes trapped in the gastro-intestinal tract Errors and safeguards in the use of long intestinal decompression tubes Swallowed long intestinal decompression tubes	
19	PARENTERAL FLUIDS AND INTESTINAL OBSTRUCTION	378
	Water metabolism Electrolyte metabolism The abnormal metabolic state produced by intestinal obstruction Parenteral fluid therapy	
20	DIVERSIONARY PROCEDURES	401
	Enterostomy Cecostomy Appendicostomy Diversionary ileocolostomy Defunctionizing transverse colostomy	
21	SURGICAL MANAGEMENT OF GASTRO-INTESTINAL OBSTRUCTION	411
	Abdominal exploration Management of obstruction of the stomach Management of obstruction of the duodenum Management of small bowel obstruction Paralytic ileus Enterostomy Bowel resection Management of small bowel carcinoma Management of large obstructed hernia Enter-enterostomy Sterilization procedure in small bowel obstruction Management of irreducible intussusception Management of obstruction of the colon	
22	PREVENTION OF ADHESIONS	426
	Avoidance of technical errors which cause adhesions Intraperitoneal instillation of substances to prevent adhesions Injections of hormones or drugs to limit adhesion formation Surgical method of preventing or selectively forming adhesions	
23	ANESTHESIA IN INTESTINAL OBSTRUCTION	443
	Local anesthesia Spinal anesthesia General anesthesia Care of patient before anesthesia Care of patient after anesthesia	
24	INTESTINAL OBSTRUCTION AFTER GASTRIC AND COLONIC SURGERY	448
	Unspecific small bowel obstruction after gastric and colonic surgery Specific small bowel obstruction following gastric surgery Specific small bowel obstruction after colonic surgery	
25	BACTERIAL TOXINS AND ANTIBACTERIAL AGENTS IN BOWEL OBSTRUCTION	477
	Role of bacteria Role of toxins Antibacterial agents in management of intestinal obstruction Dangers in the use of antibacterial agents	
26	THE EFFECT OF THE USE OF DRUGS IN BOWEL OBSTRUCTION	476
	Drugs that stimulate intestinal activity Drugs that depress intestinal activity Effect of drugs on intubation The effect of drugs on ileus The effect of drugs on mechanical intestinal obstruction	
27	DISORDERS SIMULATING GASTRO-INTESTINAL OBSTRUCTION	490
	Ruptured epigastric artery Retroperitoneal bleeding Disorders of the urinary system Coronary artery and heart disease Lesions of the spinal cord Fractured rib Acute pancreatitis and cholecystitis Diaphragmatic hernia and pneumonia Nutritional electrolyte and vitamin deficiencies Functional disorders of the small intestine and colon Unusual miscellaneous disorders	
28	NURSING TECHNIQUE IN THE MANAGEMENT OF THE OBSTRUCTED PATIENT	497
	The care of the intubated patient Care of the decompression tube Care of the suction equipment Care of the patient after surgery	
29	DEATH IN INTESTINAL OBSTRUCTION	513
	Factors affecting the mortality rate in intestinal obstruction Cause of death in intestinal obstruction	
	BIBLIOGRAPHY	525
	INDEX	543

HISTORY OF GASTRO-INTESTINAL OBSTRUCTION

ANTIQUITY

Our knowledge of intestinal obstruction dates back to antiquity. One of the earliest references to diseases of the gastro intestinal tract appears in the Code of Hammurabi written in approximately 2200 B C. The quotation which was translated by Johns is as follows: "If a doctor has cured a diseased bowel the patient shall give 5 shekels of silver to the doctor." It is probable that bowel obstruction of which fecal impaction was a common cause was among the diseases of the bowel to which this quotation referred.

The earliest surviving medical papyri those of the Middle Egyptian Kingdom (2060 to 1780 B C) claim to be copies of works written in the Old Kingdom. Authorship is even attributed to Thoth and Imhotep. Medicine appears to have been studied first in Lower Egypt. The ancient shrines of Ra Atum at Heliopolis, Neith at Sais and Anubis at Letopolis are spoken of as early centers of science.

Herodotus describes the Egyptian use of purgatives and emetics as well as their cleansing of the gastro intestinal tract for three consecutive days once a month. Since fecal impaction was not an uncommon disorder in ancient times, this cleansing operation was performed with the aid of a clyster, the precursor of the enema which was held by the Egyptians to be a gift of the Gods.

The origin of the clyster is related in Egyptian mythology. Legend has it that a party of savants walking on the banks of the Nile caught sight of Thoth in his customary form of the Ibis gravely filling his long beak with water and inserting it into his anus thereby moving his bowels. Inspired

by this, the scholars decided to duplicate the process and thus the clyster was invented. It is believed that the clyster had its earliest origin in ancient Egypt about 2000 B C.

From the earliest recorded times attempts to treat intestinal obstruction have proceeded along two main lines:

1. The indirect method where the object the relief of symptoms like distention was accomplished by using abdominal puncture to drain the intestine above the point of obstruction.

2. The use of conservative measures such as enema, cupping and bleeding.

Surgery, a third method of treatment—designed to find and remove the cause of the obstruction had not yet made its appearance.

Ileus and its treatment were referred to by Hippocrates who was born on the Island of Kos about 460 B C. However he did not make any distinction between fecal impaction and the bowel obstructions classified in later years as ileus. The method of treatment advocated by Hippocrates consisted of inflation of the intestine and the use of enemas. This remained the accepted form of treatment for a long period of time.

Praxagoras is believed to have performed the first recorded enterostomy (400 B C) in a case of severe ileus. By so doing he introduced decompression which over 20 centuries later was to play an important role in the treatment of intestinal obstruction. As early as 320 B C Praxagoras is said to have advised laparotomy in obstinate cases of obstruction.

Erasistratus who lived during the Golden Age of medicine in Greece (360 B C) subscribed to and followed the teaching of Hippocrates.

GASTRO INTESTINAL OBSTRUCTION

2

Apsyrus who lived about 300 B.C. introduced puncture of the bowel as a method of decompression. His method called for the introduction of a trochar through the abdominal wall into the distended bowel beneath and was used only in cases which failed to respond to any other measure. This operation of blind puncture through the intact abdominal wall was used to relieve distention of the abdomen due to ascites or accumulations in the intestines. This technique was later adopted by physicians and used until the latter part of the 19th century.

Celsus the most distinguished Roman medical author lived in the era of Tiberius between 14 and 37 A.D. He followed the method laid down by Hippocrates for the treatment of intestinal obstruction. With the exception of those cases of intestinal obstruction produced by fecal impaction which could be cured by enema the fate of the patient so afflicted was sealed. The outlook was so dismal that Celsus considered it one of the diseases in which the patient should be put out of his agony by the attending physician. He wrote:

In ileus it is pain that kills along with inflammation of the bowel or straining and swelling. A most acute and most disgusting form of death. For others when in a hopeless state of illness fear nothing except their impending death but those in ileus from excess of pain earnestly desire death. The physician therefore must neither be inferior to the infection nor more dilatory but if he finds inflammation to be the cause open a vein at the elbow by a large orifice so that the blood which is the pabulum of the inflammation may flow copiously and it may be carried the length of reliquum animi for or of a either the commencement of an escape from pain or of a Torpor ending in insensibility. For an ileus a breathing time though for a short space even loss of sensibility would prove an interval from pain since also to persons enduring these pains to die is happiness but to impart it is permitted when he fears that the present symptoms cannot be escaped from to lull the patient to sleep with narcotics and anesthetics.

This description by Celsus gives a very graphic idea of the ethics of the time which permitted medical practitioners to administer a toxic dose of medicine in order to relieve a suffering patient. Purges, venous section, cupping of the abdomen, immersion of the patient in warm oil or honey and most important the copious use of enemata

were recommended by Celsus for the treatment of intestinal obstruction.

16TH CENTURY

Ambrose Pare (1510-1590) was the first to recognize bowel obstruction as a pathologic entity. He reported a patient who had died from a twisted bowel. His treatment for bowel obstruction consisted of purging enemata and powdered wolf gut given orally. For severe cases he used mercury in water lead bullets smeared with mercury and a strange remedy composed of broth extract made from an old rooster who had been chased until exhausted then thrashed to death and lastly boiled with dill until the flesh fell from its bones. The prognosis for intestinal obstruction during Pare's era differed little from that reported by Celsus.

17TH CENTURY

The 17th century may well be described as the century of the enema. De Graaf (1641-1673) introduced a new flexible tube connecting the enema bag with the anal nozzle. He described this method in a treatise entitled De Chysteribus which was published in 1668. With this flexible tube which was first made of animal intestine and later of rolled and waxed leather pipe or flexible copper tubing the patient could hold the enema syringe on his abdomen and compress the piston with one hand while the other hand held the conical cannula lubricated with tallow in his rectum. The use of the enema became very popular and few homes were without one.

The hopeless prognosis associated with intestinal obstruction continued without change during the 17th century. Woodhall in 1639 described intestinal obstruction thus:

So that many which are oppressed with this disease doe perish, and dies a very miserable death ending their dates with their feces or their owne excrements issuing out of their mouths and it is many times needed for a disease infectious. In the cure of this disease no physician or chirurgeon respecting his credit will take it upon him absolutely the cure thereof especially if the death is confirmed in the patient but with pretence of death if the patient does emit the feces or excrements upwards.

Here again as late as 1639 we see the hopeless plight of the sufferer with intestinal obstruction.

which was diagnosed in that era as ileus, ilios, or iliac passion. The method of treatment advocated included numerous prescriptions of no value supplemented by the Hippocratic method of inflation enemata and blood letting.

Little or no advance in the treatment of intestinal obstruction occurred until the time of Sydenham who was born in 1624 in Dorsetshire, England. One of the greatest English physicians, he reformed the dietetic treatment in such cases and noted the great value of sedatives. Among the greatest reforms introduced by Sydenham were his writings against the use of indiscriminate enemata and vile concoctions given by mouth. However, he had not wholly given up the use of purgatives nor had he ceased using some of the methods of his predecessors. Moreover, he had great faith in the effect of a live kitten applied to the abdomen during the continuance of vomiting, and he emphasized the importance of not removing the kitten until the vomiting had ceased.

Abdominal paracentesis was used at times during the 17th century to relieve intestinal distention as well as ascites. It was referred to as a dangerous operation to be used only as a last resort. Coley was among the first to advocate the general use of this method. He performed the operation in much the same fashion as it is done today by the introduction of a trocar and cannula. When the gas had escaped, he withdrew the cannula. In the case reported by Coley, the patient, an 8-month-old infant, survived the operation but died 18 months later. In 1620 Van Helmont described a case in which the operation produced prompt subsidence of the abdominal distention, although the patient died soon after. However, the operation in this case was undertaken to relieve ascites when in actuality the patient was distended because of intestinal obstruction. Such errors were not infrequent at that time. Despite the dangers of this method, it was used rather widely until the late 19th century.

Among the earliest reports of the surgical management of intestinal obstruction was the case report of Bonetus in 1679. He described an intussusception which was successfully reduced by a young army surgeon, Paul Barbette of Amsterdam, as early as 1676, suggested opening the ab-

domen to treat intussusception and obstinate volvulus and several years later Nuck, an anatomist, supervised the successful performance of the operation in a case of intussusception.

Surgeons were becoming increasingly aware of intestinal obstruction. Many early authors described cases of intestinal obstruction caused by strangulated hernia, intussusception, volvulus, and imperforate anus. However, they did not recognize the important role adhesions and peritonitis played in obstruction.

18TH CENTURY

With the introduction of rubber in 1735, rubber enema pipes made their appearance in Europe. As a result, the use of the enema became even more widespread during this century.

Great strides were made in the 18th century in the management of intestinal obstruction. Anatomists had become familiar with the peculiarities of most of the major types of organic obstruction, although little or no progress had been made in relating these anatomic findings to the clinical picture of intestinal obstruction.

An intraperitoneal type of cecostomy was first suggested and described by Littre in 1710. Pillore of Rouen first performed this operation in 1776. He used a McBurney type of incision and sutured the open cecum to the skin. The first extraperitoneal colostomy was performed by Duret in 1793.

Renault performed the double operation of gastrotomy and enterostomy in 1772. Following this abdominal section was performed upon a small number of patients. The literature available for this century consists chiefly of case reports which deal with the various methods of treatment. The surgical approach was just beginning to emerge.

19TH CENTURY

It was not until the early part of the 19th century that fairly complete ideas concerning the pathology and symptomatology of intestinal obstruction began to appear. As a result, acute intestinal obstruction as we understand it today began to be defined and it was ultimately withdrawn from the large group of diseases simply classified as ileus. The ileus group included peritonitis, ascites, typhoid fever, fecal impaction, or anything that caused abdominal pain and distention.

ing, to crystallize. The consensus at the time was a composite of the time honored method of the ancients coupled with the more recent methods of treatment and rudimentary surgical intervention. Thinking men of the period recognizing the ineffectiveness of the older methods of treatment were beginning to cast about for newer ones.

The management of intestinal obstruction during the latter half of the 19th century was well summarized by Dujardin Beaumetz. In an excellent monograph entitled "Diseases of the Stomach and Intestines" published in 1886 the following principles of treatment were found to be in vogue:

- 1 First purgation
- 2 Metallic mercury by mouth although being favorably reported upon by many writers should be entirely disregarded from the treatment of intestinal obstruction
- 3 Interochysis with a pump or pump syringes forcing water into the colon. This method of treatment is of no value in obstructions of the small bowel
- 4 Injection of air. Hippocrates advised the employment of air for this purpose using a blacksmith's bellows of which the nozzle was introduced into the anus. At the present time it is proposed to substitute carbonic acid gas, seltzer powders, seltzer water, ibuprofen, smoke and other substances introduced into the colon in an effort to dislodge the obstructing mechanism
- 5 Intestinal puncture using a fine needle. This is generally ineffective for it is difficult to withdraw gas from the bowels by this means. After numerous such attempts Dujardin Beaumetz has hardly ever obtained a reduction in the tympanic swelling
- 6 Ice applied to the abdomen as advocated by Crisole and Masson. Application of cold to the abdomen diminishes the quantity of gas excites intestinal contraction and opposes the peritonitis which so frequently complicates strangulation
- 7 Belladonna, coffee in large doses, strychnine suspending the patient by his feet with the head downward, massage and wet cups over the whole abdomen are also useful in the treatment of intestinal obstruction
- 8 Electricity applied to the rectum. This is used in the form of galvanic current
- 9 Surgical treatment. Laparotomy is advised as a last resort. Enterostomy is a simple method but has various disadvantages. The operation for artificial anus has serious difficulties. On the one hand there is the uncertainty as to where to make the opening into the intestine and on the other the possibility of maintaining the existence of a false outlet is made at too high a point in the intestine. And then to bring the poor wretch back to life to doom him henceforth

to be the victim of a regressive and mortifying in firmly does not seem to be a very desirable result.

In this fashion Dujardin Beaumetz discussed the concept of enterostomy or colostomy as late as 1886. It would seem that these diversionary procedures were not held in very high repute at the time. The surgical approach to intestinal obstruction had been systematically worked out however. Thomas described four methods of abdominal section exclusive of surgery for incarcerated hernia which were applicable to the relief of the obstructed bowel. The methods described by Thomas are:

- 1 Colotomy which consists of incision in the left lumbar area and exteriorization of a loop of bowel usually colon
- 2 Dissection of the anterior abdominal wall and searching the abdominal cavity to correct any abnormality producing the obstruction and relieving it
- 3 Dissection of the abdominal wall and after detecting the lesion excising it
- 4 Section of the wall of the abdomen in front, seizing the first presenting portion of bowel then stitching it to the previously made incision and so incising it so as to allow its contents to escape continuously. The original cause of the obstruction being permitted to run the chance of correction by natural processes

The above principles are essentially those used today. Thomas as early as 1886 pointed out that the decision as to which surgical procedure should be adopted was not of as much practical importance as was the question of whether operative interference was necessary in the first place. Here for the first time we find emphasis being placed upon the question of surgical intervention rather than the specific procedure to be employed. The value of an exact diagnosis of intestinal obstruction was also stressed by Thomas.

20TH CENTURY

Enterostomy remained popular as a method of treatment for intestinal obstruction during the first three decades of this century. In 1902 Leiden reported five cases of postoperative obstruction cured by enterostomy alone. From this he suggested that in some cases at least a secondary operative procedure might not be necessary since the lesion producing the obstruction had corrected itself. In the ensuing years up to 1930 a number of

GASTROINTESTINAL OBSTRUCTION

technics for performing enterostomy were described

The development of the gastroduodenal tube during the early years of this century made it possible for Wangensteen in 1931 to use these tubes in the treatment of intestinal obstruction. The use of tubes marked a new era in the management of acute intestinal obstruction and as a consequence, enterostomy except in unusual circumstances has become an obsolete surgical procedure for this disorder. It was soon found that the use of a decompression tube in the upper reaches of the small intestine resulted in a considerable decrease in the mortality rate associated with intestinal obstruction. In 1938 the use of the long intestinal decompression tube and a better understanding of its function resulted in a further decrease in the mortality rate. Statistics show a reduction from the

60 per cent mortality rate reported by Gibson in 1900, to the 10 to 16 per cent currently reported by numerous surgeons throughout the country. It was soon noted, however, that misuse of the long intestinal decompression tube was responsible for some of the deaths.

In recent years the widespread use of antibiotics, better anesthesia, the availability of blood, an improved knowledge of the role of the long intestinal decompression tube, and the introduction and use of the intestinal decompression sound have resulted in a further reduction in the mortality rate. In some centers a mortality rate of 5 per cent or less is being achieved. The education of the public as well as physicians in the value of earlier diagnosis and earlier surgical intervention in cases of acute mechanical intestinal obstruction has also contributed to the reduction in these rates.

EMBRYOLOGY OF THE GASTRO- INTESTINAL TRACT

Gastro intestinal obstruction is not infrequently the result of disturbances in the normal embryologic development. It has long been known that organic obstructions of the bowel occur at the sites of the most complex embryologic processes. The areas most commonly involved are

- 1 The esophagus at the level of the bifurcation of the trachea
- 2 The duodenum close to the ampulla of Vater
- 3 The lower ileum in the area of the vitelline duct
- 4 The rectum
- 5 The anus

Although these are the most common sites of obstruction due to deranged embryology, intestinal obstruction may occur anywhere along the gastrointestinal tract. If one understands the mechanism involved in the developing embryo, the problems of intestinal obstruction based upon such derangements become simple. For purposes of clarification in this chapter we may oversimplify these embryologic processes without entering into some of the conflicting concepts held by students of embryology.

ESOPHAGUS

Normal Variations

The esophagus makes its appearance at about the fifth week of fetal life. It can be recognized at that time as a short tube extending from the pharynx to the stomach. As the neck and growing heart differentiate, the esophagus elongates so that in a 5½ week embryo (9 mm) it is well defined. The 6 week embryo presents an esophageal lining

epithelium two layers thick. Vacuoles appear in the lining epithelium at about the eighth week of development. As a result the lumen becomes channelled but not occluded as does the duodenum. At birth the lining epithelium is 10 cell layers thick. The superficial glands make their appearance at four months, the deep glands appearing later in fetal life. Because the esophagus remains attached to the dorsal wall by a broad mesentery and never develops a mesentery of its own, it remains bare of serosal covering over its posterior wall.

Anomalies

The most commonly found anomalies obstructing the esophagus are the result of a persistence of the partial epithelial occlusion found at the eighth week of fetal life. If the occlusion is partial, a stenosis results. In some instances the occlusion becomes complete, resulting in an atresia which is not infrequently associated with fistula into the trachea. The incidence of esophageal atresia has not been definitely established. It is thought to be extremely rare. Shukowsky reported only one case in 50,000 newborns. Haight reported an incidence of esophageal atresia of approximately 1 in 2000 births. In general, esophageal atresias fall into three groups:

- 1 Complete absence of the esophagus or presence of a solid cord
- 2 Presence of an upper and lower segment each ending in a blind pouch
- 3 Cases with a fistula joining the esophageal segment with the trachea or bronchus. In this group the fistula may communicate between the

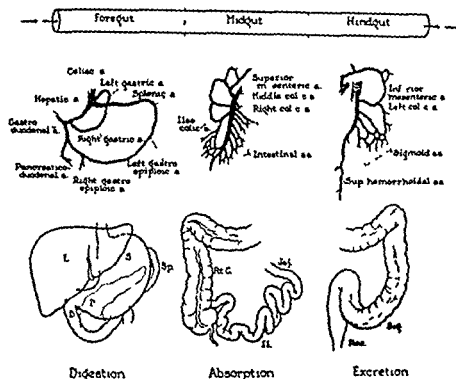


FIG. 1 Note that the midgut is the anlage for the entire small bowel and half of the colon

upper segment and the trachea or between the lower segment and the trachea or bronchus or in another variety there may be a fistula between both segments and the trachea or bronchus. It has been reported that 80 per cent of the esophageal atresias with tracheo esophageal fistulas occur with the fistula between the lower segment and the trachea or bronchus.

STOMACH

Normal Variations

The gastro intestinal tube which consists of an internal tube of endoderm and a covering layer of splanchnic mesoderm is recognizable at the fifth week of embryologic development the foregut and hindgut having made their appearance one week earlier. The covering layer of splanchnic mesoderm becomes the gastro-intestinal musculature connective tissue and covering peritoneum. The endoderm is the primary gastro intestinal tube which develops into the epithelial lining gland and glandular outgrowths. Of the two muscle layers the inner circular layer develops at the seventh week whereas the longitudinal layer is not recognizable until the twelfth week.

In a 5 week embryo the stomach appears as a spindle shaped swelling of the foregut. It origi-

nally is found at the level of the heart at a relatively high point. By the end of the seventh week it is found much lower in the region it is to occupy at birth. During this period of time the stomach changes markedly in size as well as in shape and position. Whereas in a 5 week embryo it is found to be a fusiform bulge, in a 6 week embryo it has already begun to take on the funnel shape. At the end of the seventh week the stomach may be readily recognizable as the adult configuration.

During this period from the fifth to the eighth week of fetal life the stomach undergoes many changes. The dorsal border grows faster than does the ventral border with the result that the convex greater curvature and the concave lesser curvature become recognizable. The fundus then arises as a local outpouching near the superior end. At this time the stomach rotates on its long axis approximately 90 degrees. By so doing the greater curvature lies to the left and the lesser curvature to the right. The original right and left surface of the stomach with the corresponding vagus nerve become as a result of this rotation dorsal and ventral. Therefore the left vagus nerve is found on the ventral surface and the right vagus on the

dorsal surface of the stomach. During this period of rotation the enlarging liver pushes the freely movable superior portion of the stomach to the left. The inferior end of the stomach, being relatively fixed by the ventral mesentery and the bile ducts, changes little in position. As a result the stomach stretches across the abdomen from right to left with the fundus lying under the left leaf of the diaphragm.

Mucosal pits are recognizable in the stomach of a 7 week embryo. At 14 weeks gastric glands begin to differentiate from the pits. These multiply remarkably so that at birth 14 000 000 glands are said to be present.

Anomalies

As a result of an arrest in development the stomach may be found above the diaphragm. It may be found on the right side of the abdomen. The most common anomalies of the stomach are stenotic and most frequently involve the pylorus with an incidence of 1 in every 100 births reported. Gould and Pale refer to a dissection in which the stomach was found to be completely absent. There are many reports of two stomachs being found in which two actual cavities could be identified. In addition to this type instances of hour glass stomach have been reported in which the constriction at the middle was so marked that a duplex stomach resulted. Crooks reported a case in which the stomach of an infant terminated in a cul de sac.

THE DUODENUM AND PANCREAS

The first portion of the duodenum is carried with the stomach during its period of growth and torsion. As a result the duodenum assumes a U shape when the stomach assumes its transverse position. This is further accentuated by the development of a pancreatic bud within this curve. The shortened mesentery of the duodenum fixes it to the posterior abdominal wall. Because the development of the duodenum is controlled by intra-abdominal pressures its position is generally constant. The duodenum up to the ampulla of Vater is derived from foregut.

Ventral Mesentery

A persistence of a portion of the ventral mesentery produces a fold which may cause compres-



FIG. 2. An example of reversed rotation of the first limb of the duodenum.

sion of the duodenum at the junction of its first and second portions. As the duodenum undergoes rotation this persistent portion of the ventral mesentery could fix, compress or kink a segment of duodenum. This might result in stenosis. Normally the falciform ligament of the liver and the anterior wall of the omental bursa represent the persistent portion of the ventral mesentery. The anterior wall of the omental bursa is divided into the gastrohepatic and the hepatoduodenal ligaments. These are the remains of the ventral mesentery. The hepatoduodenal ligament usually ends caudally at the epiploic foramen. If the ligament extends caudally beyond this point it produces a fold resulting at times in duodenal obstruction. This is the hepatoduodenocolic band.

Pancreas

The development of the pancreas may be noted as two outpouchings from the duodenal enterodermal lining in a 4 mm (4½ week) embryo. One bud pushes out from the dorsal wall into the dorsal mesentery just proximal to the hepatic diverticulum. This dorsal bud grows across the body toward the left until it reaches the spleen. This gives rise to the body, tail and ventral portion of the head of the adult pancreas. The ventral pancreatic bud appears in the angle between the bowel

and the hepatic diverticulum. It may be more or less bilobed. A left lobe and a right lobe are recognizable. The ventral bud remains smaller than the dorsal and a short ventral duct is carried away from the duodenum by the lengthening common bile duct from which it arises directly. Unequal growth of the duodenal wall with rotation of the stomach and duodenum and elongation of the common bile duct to the right brings the ventral lobe of the pancreas up into the dorsal mesentery to approach and ultimately fuse with the dorsal pancreas. The ventral pancreas then forms the dorsal portion of the head and part of the uncinate process of the pancreas. The tip of the ventral pancreatic duct unites and anastomoses with the main stem of the dorsal duct. This united duct is the main pancreatic duct of Wirsung which empties into the duodenum at the ampulla of Vater beside the common duct or into the common duct. The remaining proximal portion of the dorsal pancreatic duct may atrophy. At times it persists and may open into the duodenum above the ampulla of Vater and is recognized as the duct of Santorini.

Pancreatic and Duodenal Anomalies

The anomaly of an annular ring of pancreatic tissue is the persistence of the left half of the ventral inflex. The tip may remain fixed to the duodenal wall in an anterior position and become stretched around the right side of the duodenum upon migration of the remaining portion of the ventral pancreas. This lesion of annular pancreas producing duodenal obstruction is relatively uncommon. Mesenteric may involve any portion of the duodenum as a result of an arrest in development in the prevascularization phase of duodenal lumen development. An arrest in the phase of coarctation of the vitellines by means of which a lumen develops within the duodenum may result in a marked narrowing of the duodenal lumen. This is a stenosis of the duodenum. This is variable in degree from a high grade partial obstruction to the low grade obstructive process found in duodenal diaphragm formation. Duplication may occur along the duodenum. This may communicate with the intestinal lumen or may present itself as a cystic mass producing obstruction. Ladd demon-

strated a case of this type. A reversal in direction of the first limb may occasionally occur.

THE SMALL INTESTINE AND COLON Development of the Intestinal Canal

In the youngest recognizable embryo (a 5 week embryo) the intestinal tract is found as a simple tube which begins at the stomach and ends at the cloaca. The segments of intestine are indicated as cephalic and caudal limbs of the intestinal loop with the point of origin of the yolk stalk marking the dividing point. The intestinal canal is supported from the dorsal body wall by the dorsal mesentery.

In the fifth to the sixth week of embryonic life the ventral flexing of the intestinal loop becomes quite marked. At this time the attachment of the yolk stalk to it disappears. In addition a bulging appears in the caudal limb at the site of the future cecum. This then marks the boundary between the small intestine and the colon. At this time three distinct loops are recognizable. There are the foregut, the midgut and the hindgut. They are attached to the ventral and to the posterior abdominal wall in the midbody line by their respective mesentery. In each mesentery, the specific vascular supply to the organ to which it is attached is found. The celiac axis is found in the mesentery of the foregut, the superior mesenteric axis in the mesentery of the midgut and the inferior mesenteric artery in the mesentery of the hindgut.

The foregut and the hindgut remain relatively fixed in position. The midgut however undergoes remarkable developmental changes. The midgut loop so increases in size that the abdomen can no longer hold it. As a result it protrudes into the umbilical cord at about the seventh week of embryonic life. Just prior to this protrusion the midgut loop undergoes counter clockwise rotation with the superior mesenteric artery as its axis. By this rotation the previous cephalic limb is carried from the midline to the right and caudally through an arc of 140 degrees. The superior mesenteric artery is found to enter the mesentery of the midgut at the constricted area between the duodenum and the celiac angle. This narrow mesentery is known as the fetal mesentery. The first stage of rotation which has been described in

EMBRYOLOGY OF THE GASTROINTESTINAL TRACT

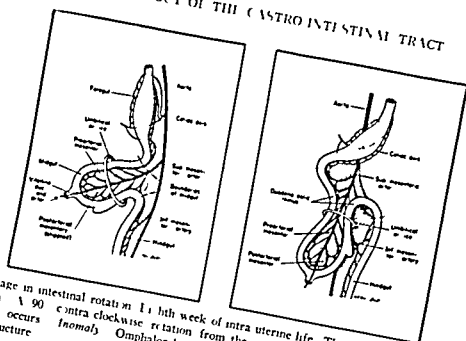


FIG. 3 First stage in intestinal rotation. Fifth week of intra uterine life. The midgut loop is within a temporary umbilical hernia. A 90° counter clockwise rotation from the sagittal (left view above) to the horizontal plane (right view above) occurs. *Anomaly* Omphalocele Arrest of rotation with a persistent umbilical hernia covered by jelly like cord structure

thought to be due to the downward pressure of the persistent umbilical vein upon the pre arterial loop of the midgut. This occurs as a result of the downward pressure upon the left umbilical vein by the rapidly enlarging liver. The post arterial limb of the midgut is consequently pushed up and to the left. This protrusion of the midgut loop into the umbilical cord constitutes a normal hernia. This whole first stage of rotation is then completed by the tenth week of fetal life.

Return to Peritoneal Cavity

By the tenth week of fetal life the abdomen has increased sufficiently in size to permit return of the herniated loop of bowel. This marks the beginning of the second stage of intestinal rotation. At this time a definite shrinkage of the liver has occurred. As a result of pressure changes within the abdominal cavity the midgut gradually begins to return to the abdomen. It does this in an orderly fashion and rather suddenly. The small intestine returns first. It passes behind the tightly stretched mesenteric artery which passes obliquely downward from its origin at the aorta out to the umbilical opening. The first portion of the small bowel passes counter-clockwise from right to left. Other loops follow until all have entered except the cecum with the termination of the superior

mesenteric artery. As the midgut passes into the abdomen its coils pass beneath the superior mesenteric artery and crowd over to the left and upper corners. As a result the hindgut is pushed from its midline position over to the left and downward and backward. The first coil to leave the umbilicus carries the terminal end of the superior mesenteric artery with it. The cecum follows but comes to be anterior to the small bowel and the mesenteric artery on a level with the umbilicus. The cause of the withdrawal is controversial. The various factors proposed are (1) the sucking effect of a decreasing tension within the abdomen as a result of a decline in the rate of growth of the liver as compared with that of the abdominal cavity (2) the pull of the nonherniated bowel (3) the retraction from the slower growing mesentery. As the colon grows it lengthens and straightens out carrying the cecum upward and toward the under surface of the right lobe of the liver. This completes the second stage of rotation.

As the colon elongates it passes downward to the right iliac fossa carrying with it the mesentery and the superior mesenteric artery. In this third stage of rotation the cecum assumes its adult position. The post arterial mesenteric leaflet becomes obliterated and the pre arterial leaflet fuses with the posterior parietal peritoneum to form the

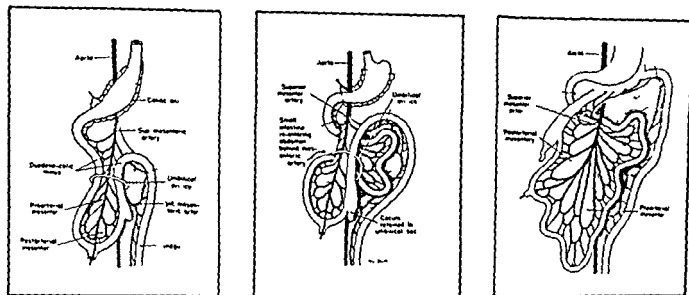


FIG. 4. Second stage in intestinal rotation. Tenth week of intra-uterine life. The proximal limb of the midgut loop re-enters the celomic cavity under the mesenteric root first (*center view*). An orderly reduction of the remaining midgut loop occurs. As the cecum and ascending colon are reduced they straighten out (*right view*) completing an 180° contra-clockwise rotation of the midgut above the mesenteric root. The duodenum is thus drawn under and the colon above this vascular pedicle. *Anomalies:* Nonrotation, volvulus of midgut, malrotation, reversed rotation.

mesentery of the small bowel. This mesentery becomes fixed to the posterior abdominal wall from the second lumbar vertebra on the left downward to the right three vessels. The fusion with the cecum and ascending colon is so marked that the mesentery is obliterated. The mesentery of the transverse colon, however, remains long. Descent of the cecum and ascending colon is usually completed by the eighth month of fetal life. The cecum has now attained its adult position. Fixation proceeds thereafter. The final peritoneal relationships, however, are not established fully until four months after birth.

The possibility of hypofixation is balanced by that of hyperfixation. In this event the process goes to excess, resulting in an entirely retroperitoneal cecum. This may at times involve the terminal ileum which also becomes retroperitoneal. Hyperfixation may be local; the principle sites being the beginning of the transverse colon and the hepatic flexure. A point of local hyperfixation combined with looseness of the proximal large bowel provides an intimate situation favorable for a cecal volvulus. Lickstein's membrane, Lanes' band, and various peritoneal membranes may be explained as congenital aberrations at this period in development.

The descending colon is pushed again to the body wall to the left. It loses its mesentery. It is originally thinner than the small intestine and does not become greater in diameter until after the fifth month.

Anomalies

Meckel's diverticulum is formed by an endodermal-lined yolk stalk which detaches from the permanent bowel during the sixth week of fetal life and soon degenerates. When the proximal end of the yolk stalk persists a Meckel's diverticulum results. This usually occurs on the antimesenteric border of the distal third of the ileum. It rarely originates in the appendix. The diverticulum may extend to the umbilicus as a continuous channel or a cord. Up to 10% approximately 151 cases of patent omphaloenteric duct have been reported. Intestinal obstruction may result from Meckel's bands.

Any deviation from the orderly rotation or re-turn of the midgut to the abdominal cavity may result in gastrointestinal anatomic disturbance resulting in obstruction. The foregut and hindgut, because of their constant development and because their development is not as complicated as that of the midgut, are uncommonly involved in

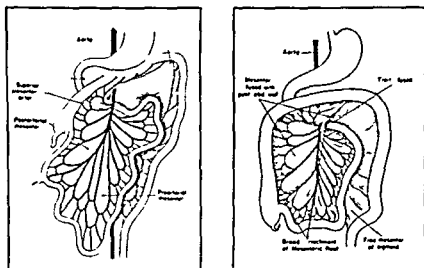


FIG. 2 Third stage in intestinal rotation. Completed at birth. Descent and fixation of the cecum from right upper quadrant (left view above) to right iliac fossa (right view above) occurs. The mesentery of the small intestine acquires a broad attachment from Treitz ligament to right iliac fossa. *Anomalies:* Subhepatic cecum, mobile cecum, retrocecal appendix.

anomalies of nonrotation, malrotation, or reverse rotation. Most of the disturbances in rotation of the midgut are found in the second stage of rotation. Dott explains abnormalities in rotation of the midgut as being due to the fact that the duodenocolic angle is widened in some instances. As a result the orderly and gradual return of the prearterial loop of midgut to the abdomen does not occur. Pathologic derangements of the midgut depend upon which direction the cecum takes at its reduction to the abdomen. In nonrotation it may pass upward into the left upper quadrant so that the entire colon and cecum are arranged to the left of the midline with the entire small bowel to the right of the midline, the ileum entering the cecum from right to left. In malrotation the cecum passes up to the region of the pylorus and becomes fixed there. At times it may remain in the subhepatic area. In these cases there is an elongated and narrow mesentery of the fetal type with no point of fixation of the ileum and jejunum except at the narrow duodenocolic angle. In reverse rotation the loop of small bowel instead of passing from right to left behind the superior mesenteric artery in a counter clockwise manner passes from left to right in a clockwise manner. As a result the cecum comes to lie behind the

superior mesenteric artery where it is fixed. This anomaly is rare.

Derangements of the third stage consist largely of undescended cecum with fixation in the subhepatic area or a pelvic cecum due to nonfixation.

ANUS AND RECTUM

The terminal portion of the colon is derived by the horizontal division of the cloaca. The human cloaca recapitulates the stages in development found in lower forms of life. The cloaca in humans is divided by the cloacal septum into a dorsal rectum and a ventral urogenital sinus. This division is completed by the seventh week of embryonic life. At this time the cloacal membrane ruptures. This exposes the tip of the down growing cloacal septum which is covered with endoderm and becomes the perineum. After the cloacal membrane ruptures a short ectodermal proctodeum meets the endodermal rectum to form the anal canal.

There are four major types of rectal abnormalities:

1. All conditions in which there is an imperforate anus due to a membrane.
2. A patent anus is present but there is stenosis of the rectum at a higher level.

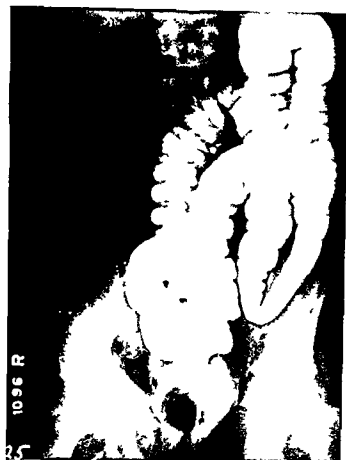


FIG 6 Partial nonrotation of the bowel



FIG 7 Failure of intestinal rotation. Note the cecum on the left side of the abdomen and the terminal ileum entering the cecum from the right

3 The anus sphincter and lower portion of the rectum are normal but the upper portion of the rectum ends blindly above the lower pouch.

4 The anus is imperforate and the rectal pouch ends blindly some distance above the anus.

In addition to the obstructive types of anorectal anomalies various types of fistulas resulting from embryologic derangements may occur between the rectum, vagina, bladder, perineum or urethra. These anomalies may occur alone or together. Such anomalies are said to occur about once in every 1,000 newborn.

INTESTINAL LUMEN

Prior to the fifth week of fetal life the intestinal tract presents a well defined rounded lumen lined with endodermal epithelium. Soon after this the epithelium rapidly proliferates and the lumen of the intestine becomes obliterated by epithelial confluences. This solid stage persists for a short time until vacuoles appear and coalesce to reestablish the intestinal lumen by the twelfth week

of embryonic life. An arrest in development during this period results either in atresia of the intestine at one or more points or in a stenosis. The atresia is due either to complete lack of continuity with a blind end or to the remaining of an imperforate septum. In cases of atresia the intestine below the point of obstruction appears as a smooth cord although it has all its normal elements including a lumen. Generally this is only large enough to admit a fine probe. In stenosis the intestine below the point of obstruction remains small but not as small as in atresia. In rare instances with complete lack of continuity of the bowel multiple blind ends may be found. Although the process of epithelial exclusion and subsequent vacuolization normally takes place primarily in the duodenum an appreciable percentage occurs in the terminal ileum. The process of vacuolization also occurs normally in the esophagus yet esophageal atresias are seldom found in this area.

The case in which there is complete disruption of intestinal continuity with a mesenteric defect in addition cannot be well explained by the hypothesis of coalescence of vacuoles within the gut lumen. The incidence of atresia is reported as 1 in 20 000 births.

Duplication of the small bowel may occur as an arrest in development during this stage of coalescence. This may take the form of a blind pouch with no opening into the normal small bowel or it may be attached at the proximal end distal end or both. These duplications usually occur on the mesenteric side of the bowel.

MYENTERIC PLEXUSES AND GANGLIA

The ganglia of the trunk develop from the sympathetic primordia present in the vicinity of the notochord throughout its length. These can be noted in the 5 mm embryo (4½ weeks). The primordial cells migrate outward before differentiation of nerve elements has occurred. The ganglion cells cluster in masses during the seventh week. These ganglionic masses are then joined together on each side by a longitudinal nerve cord. These become the sympathetic trunks. Aggregates of primordial nerve cells migrate more distally to form the prevertebral plexuses such as the celiac. These may be seen at the eighth week of embryonic life. The myenteric plexuses of Auerbach and Meissner are still more distally placed. Each cell in these terminally placed myenteric plexuses is in direct relation with the axon of a cerebrospinal cell. The plexuses of Auerbach and Meissner may be noted at about the eighth week of development. The cells of the terminal myenteric plexuses are thought to advance peripherally along the vagus nerves although an origin from the prevertebral ganglia has been suggested.

Adynamic obstruction may occur as a result of a congenital absence of ganglionic elements in the sigmoid or rectum. This results in a tremendous megacolon. Occasionally these same aganglionic areas are noted in the terminal ileum. This defect was first described by Hirschsprung in 1886.

MESENTERIES

The primitive mesentery is formed by the splanchnic mesoderm which overlies the endoderm

swinging inward from each side toward the midplane. This forms a double layered partition which contains the gastrointestinal tract between its layers. The bowel itself at this time consists of a straight tube. The bowel divides the mesentery into two halves, the anterior half called the ventral mesentery and the posterior half the dorsal mesentery. Soon this region of attachment becomes relatively narrower and the bowel is then suspended throughout most of its length by a definite dorsal mesentery which extends like a curtain in the midplane. The pharynx and upper esophagus have no mesentery except at the very beginning. The lower esophagus lies in a specialized portion of the mesentery which is designated as the mesoesophagus or mediastinum. However the gastrointestinal tract within the peritoneal cavity is attached by a dorsal mesentery to the posterior abdominal wall. The mesentery is further classified as mesocolon, mesosigmoid, mesogastrium—the name corresponding to the structure or organ which it supports.

As long as the bowel was a straight tube the dorsal mesentery was a simple sheet whose attached edges were of equal length. Since the intestine becomes elongated at a faster rate than the abdominal wall the posterior portion of the mesentery fixed to the abdominal wall becomes much shorter than the intestinal attachment of the mesentery which grows in length with the bowel. With intestinal rotation upon the return of the midgut to the abdominal cavity the transverse colon is carried anterior to the duodenum and the small bowel lies caudal to the ascending colon and to its left. There is thus a torsion of the mesentery with the origin of the superior mesenteric artery as an axis. Using the point of origin of the superior mesenteric artery as it emerges from below the duodenum as a focal point the mesentery of the small bowel and colon spreads out in a funnel shape.

By the fourth month the intestine which was previously freely movable within the range of its attached mesentery develops secondary fusions. These fix bowel segments and produce new lines of attachment. These appear to be related to the erect position assumed by man. These secondary developmental adhesions are formed by a process similar to that following inflammation. The right

and left colon are fixed to the right and left sides of the abdomen respectively prior to mesenteric fixation. The flat surfaces of their mesentery lying flat against the posterior abdominal wall fuse with the covering peritoneum and remain permanently fixed. The transverse colon, however, which has remained relatively free, now becomes fixed by a new transverse line of origin (transverse mesocolon) which fuses at its roots with the anterior surface of the duodenum and pancreas. The mesocolon presses duodenum against the posterior body wall and obliterates its mesentery. The pancreas being an outgrowth of ectoderm of the gut into the mesoduodenum, also assumes a retroperitoneal position behind the root of the mesocolon. The mesentery of the small intestine normally does not form secondary attachments nor does the mesosigmoid.

The ventral mesentery is formed by a continuation around the primitive gut by the same splanchnic mesodermal layers that formed the dorsal mesentery. The ventral mesentery in its abdominal portion is concerned with the development of the liver. It grows downward and forward into the ventral mesogastrium and splits the component leaves apart thus ensheathing it with the same mesenteric relations as the heart. As the liver increases in size within the sheath of the ventral mesogastrium, the capsule and ligaments are formed. That area of the liver in apposition to the diaphragm remains bare of covering. The enveloping layers then form the capsule of the liver which is continuous with the peritoneum of the abdominal cavity. The liver maintains its connection permanently with the ventral mesentery along its mid dorsal and midventral lines. That portion of the mesentery between the liver, the stomach and the duodenum is the lesser omentum which is further divided into hepatogastric and hepatoduodenal ligaments. The attachment of the mesentery of the liver to the anterior abdominal wall becomes the falciform ligament. Caudal to the duodenum the ventral mesentery does not exist.

Variations in form and relation are quite frequent in the development of the mesentery. In general, these consist of the persistence of fetal types of mesentery. During the course of development of the various mesenteric abnormal openings or

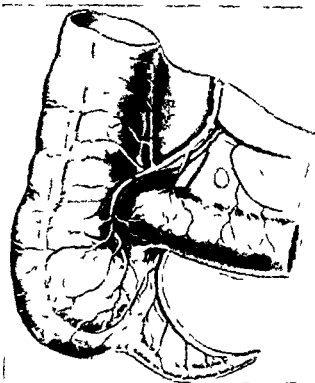


FIG. 8 Note the opening at the ileocecal mesenteric angle. Note the blood supply at this point. The point of opening is relatively avascular. The opening is distal to the terminal small intestinal vessels but it is proximal to that supplying the ileocecal junction, the appendix and the proximal cecum.

slits may be found. These occur during the developmental period in intra uterine life and are most frequently located in the mesentery to the ileum within 2 to 3 inches of its junction with the cecum. Opening in the mesentery of the right colon and the sigmoid as well as the greater omentum have been found less frequently. The importance of the openings lies in the occasional herniation of a loop of bowel with a resultant obstruction. The most serious defect in the secondary mesenteric attachment has been called the enterium commune. In this condition the entire small and proximal half of the large bowel are suspended from a midline pedicle which is attached to the posterior abdominal wall at the level of the superior mesenteric artery. Although this simplification of the mesentery is not incompatible with health, it predisposes to massive midgut volvulus. The inception of volvulus in such cases may be either prenatal or postnatal. Occasionally a case is reported in the adult. The entire jejunum

ileum and proximal portion of the colon twist in a clockwise or counter clockwise direction. In such cases the terminal duodenum and midtransverse colon become obstructed. A massive volvulus of the intestine in the enterium commune may simulate reversed intestinal rotation. Reversed intestinal rotation has its inception at the tenth week of fetal life and when it occurs further intestinal and mesenteric development is affected. The reversed rotation would be normal for that specific individual. In this type of case surgical manipulation designed to return the bowel to the normal position for man would result in a volvulus in this specific type of individual. In order to distinguish between midgut volvulus, malrotation and reversed rotation the entire bowel must be exteriorized so that the relationships are established.

BURSA

The omental bursa or lesser peritoneal cavity is developed in the dorsal mesogastrium. This may first be noted in a 4 week embryo as a peritoneal pocket which passes cephalically into the thick dorsal mesentery. It passes to the right of the esophagus and as far cranially as the right lung bud. The apical end of the sac becomes constricted off as a small closed cavity called the infracardiac bursa. This may persist in the adult or disappear. A similar pocket on the left side disappears shortly after four weeks. Two other clefts tend to separate the liver and pancreas from the bowel. These clefts unite into one cavity. This represents the beginning of the omental bursa. At $\frac{1}{2}$ to 6 weeks (8 mm embryo) the caval mesentery can be noted as a ridge of the mesentery which continues caudally along the dorsal body wall. In this the inferior vena cava develops. The expanding liver comes into relation with this caval mesentery

and rapidly grows caudally so that with the caval mesentery it forms a partition between the peritoneal cavity and the omental bursa. In the 5 week embryo the omental bursa is crescent shaped and is bounded medially by the dorsal mesogastrium and the right wall of the stomach. Laterally by the right lobe of the liver and the caval mesentery. The epiploic foramen is the aperture through which the lesser and the main peritoneal cavities communicate.

With the rotation of the stomach and the increase in size of the lesser sac the epiploic foramen becomes quite small. The foramen becomes bounded ventrally by the edge of the lesser omentum dorsally by the inferior vena cava cranially by the caudate lobe of the liver and caudally by the wall of the duodenum. At this time due to the rotational process the lesser peritoneal cavity becomes a flattened sac in its dorso-ventral diameter.

In the third month (68 mm) of embryonic life the flat dorsal wall of the sac fuses with the dorsal body wall. When this process of fusion reaches the transverse colon it fuses where it lies upon the colon and mesocolon. As a result the transverse mesocolon becomes four layers thick. The omental connection between the stomach and colon becomes the gastrocolic omentum. Caudal to this the walls of the omental bursa unite and obliterate the cavity.

The small bowel may herniate into the lesser omental bursa because of an abnormally large foramen. A similar occurrence may take place in an abnormally capacious duodenojejunal or ileocolic peritoneal recess. The incarceration of a large part of the small intestine within the lesser peritoneal cavity has been attributed in several instances to reversed fetal rotation.

ANATOMY OF THE GASTRO- INTESTINAL TRACT

In its broadest sense the gastro-intestinal tract which is concerned with the ingestion, utilization and transport of food must be considered as beginning with the mouth and ending at the anus. We are not accustomed to think of the nasopharynx and esophagus as part of the gastro-intestinal tract; the very name would suggest that it begins at the stomach and ends in the colon. Nonetheless, in any discussion of intestinal obstruction one must consider this tract in its broadest sense. Although obstructions of the mouth and nasopharynx are extremely uncommon causes of intestinal obstruction, the treatment of the obstructed bowel by means of the long intestinal decompression tube presupposes an accurate knowledge of the anatomy of the nasopharynx as well as the esophagus, stomach, small bowel and colon. For this reason we include the anatomy of these structures in this section.

NASOPHARYNX

The nasal cavity is divided by the septum into a right and left half. Each of these may be spoken of as the cavity of the nose. Each cavity is between 5 and 7 cm. long, but is narrow in width with the widest point at the floor where it is 1 cm. wide and the narrowest point at the roof where it is only 2 mm. wide. The width is further reduced in size by the conchae which project into the cavity from the lateral wall. These in turn are covered by cavernous tissue with remarkable powers of expansion and contraction depending upon the degree of engorgement.

The external opening of the nose is the nostril

which face downward. The internal opening faces downward and backward into the pharynx. Although the nasal cavity would seem to be directed upward and backward, the insertion of a tube into the nose would show that the nasal cavity is actually directed downward and backward. The cavity is higher in the front and slopes gently backward.

The conchae are three curled bony plates that project into the nasal cavity and divide it into three spaces: (1) inferior meatus, (2) middle meatus, and (3) superior meatus. The middle meatus is generally much more roomy than the superior or inferior meatus. The conchae are covered with cavernous tissue which has the capacity to swell greatly, reducing the size of the middle and inferior meatus.

There are wide variations in the normal nose with regard to the width of its passageway. An individual with a broad nose will present a spacious channel for the passage of the long intestinal decompression tube, whereas one with a very narrow nose may present a passageway so small that it requires considerable ingenuity to pass a tube. Between the two extremes there are all gradations in width.

In the nasal passage there are many deviations from the normal which must be watched for. Enlargement of the turbinates, whether due to congestion based upon allergy or upon inflammation, may so narrow the nasal passage that only the smallest caliber tube will pass. In such cases the liberal use of nasal decongestants usually reduces the turbinates enough to create an adequate pas-

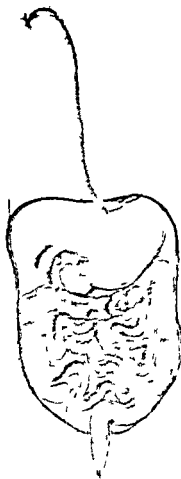


FIG. 9 Diagrammatic representation of the normal gastrointestinal tract. The small bowel has been shortened for convenience of illustration.

sage for a 16 Fr. tube. It is surprising how large the inferior or middle meatus becomes when the engorged turbinates are adequately shrunk.

The floor of the nose may protrude upward creating another variation from the normal. This results in such narrowing of the inferior meatus that it becomes unsuitable for the passage of an intestinal tube. In such cases the middle meatus may be used. It must be borne in mind also that the direction of this passageway is downward and backward and therefore the mercury weighted intestinal tube must be gently pushed back into the nasopharynx in this direction.

The presence of a deviated septum may result in such narrowing of the side of the nose toward

which the septum is bent that the passage of any tube through that side is difficult or impossible. In addition the opposite nasal passage may present considerable narrowing due to the congestion and hypertrophy of the turbinates so often associated with a deviated septum. The usual finding in these obstructive cases is chronic sinusitis. Nasal polyps are apt to be a concomitant finding.

The passage of an intestinal tube through a passageway covered with inflamed and congested mucosa and narrowed by deviation of the septum requires not only considerable skill but also a good measure of tact. To pass a long intestinal tube under such conditions without pain and with a minimum of discomfort and to do so without rendering the patient uncooperative and annoyed one must utilize his knowledge of the anatomy of the nose.

The nasopharynx is rarely a barrier to intestinal intubation but obstructions in this area are not uncommon. Hypertrophied adenoid tissue in children may so encroach upon this passage as to render the passage of a nasogastric tube difficult. Retropharyngeal infections, tumors at the base of the skull such as a chordoma and neoplasms of the nasopharynx may also cause obstruction. Such obstructing processes rarely interfere with alimentation. Their association with an obstruction in the small bowel however might obviate the passage of an intestinal decompression tube. In such cases early surgery using the intestinal decompression sound or transoral intubation would be required.

The oropharynx rarely is the seat of obstructive processes. Occasionally the necessity of intubating a patient with paralysis of the muscles of deglutition may create a problem. One of the earliest cases of this type appeared in 1790 in a report by John Hunter entitled *A Case of Paralysis of the Muscles of Deglutition Cured by an Artificial Mode of Conveying Food and Medicines into the Stomach*. In this Hunter described the use of a tube introduced by a stylet of whalebone. In the introduction of the mercury weighted intestinal decompression tube we depend upon the action of the pharyngeal constrictors to carry the tube through the oropharynx into the esophagus. The use of a stylet might be required to introduce such tubes in the event of paralysis of pharyngeal con-

of insertion of the phrenicoesophageal membrane to the esophagogastric junction has been the source of considerable contradictory anatomic study. Opinion is equally divided as to the presence or absence of a sphincter at the esophagogastric junction. The consensus at present seems to indicate that although no anatomically distinct sphincter muscle is demonstrable nevertheless a sphincter mechanism can be demonstrated. Experimental studies have shown in addition that the sphincter mechanism can function independently of the esophageal musculature. The importance of this mechanism in obstructions of the lower esophagus cannot be overemphasized. Achylasia may so completely obstruct the esophagus at this point that surgical intervention in the form of gastrostomy is required to maintain nutrition. Cardiospasm due to fear, emotional upset and trauma to the nose in the process of nasogastric intubation creates a real barrier to the passage of an intestinal tube.

The physiologic constrictions of the esophagus are (1) just below its origin at the cricoid cartilage (2) just above the crossing of the left bronchus (3) proximal to the esophageal ampulla from 2 to 6 cm. above the diaphragm and (4) infradiaphragmatic constriction of the esophagus from pressure of the liver. Contrary to general belief the esophagus is not physiologically constricted at the diaphragmatic hiatus.

The mucosa of the esophagus is arranged in longitudinal folds. They generally pass downward in a parallel fashion to the lower esophagus where they twist in a spiral. This lower twist has been explained as being the result of torsion of the stomach from its embryonic midplane position to its transverse position. The lamina propria mucosa is a thin fibrous layer that separates the epithelium from the muscularis mucosa. This latter is a well developed muscular layer in the lower portion of the esophagus.

The muscular portion of the esophagus consists of an inner circular muscle and an outer longitudinal muscle. These are separated by a well defined layer of connective tissue. Of the two the inner circular muscle is the thickest and the most developed. The upper portion of the esophagus presents striated muscle which merges at about its



FIG. 10 Normal stomach upright position in an asthenic individual (a) Gas bubble at the cardiac end (b) Inferior pole of stomach in this position (c) Incisura angularis (d) Pyloric canal markedly contracted (e) Normal duodenal bulb (f) Second portion of duodenum (g) Hepatic flexure of colon

midpoint into smooth muscle. The downward propulsion of solid food depends upon peristaltic waves in these muscles.

STOMACH

The stomach is the most dilated portion of the digestive tube. It begins at the esophagogastric opening to the left of the midline and extends obliquely and inferiorly across the abdomen to end at the duodenum to the right of the midline. It is flattened in its antero-posterior diameter so that it presents two surfaces—a dorsal and a ventral. In addition it presents two curvatures—a lesser concave curvature facing cephalically and a greater curvature which is convex facing inferiorly. The gastrophilic omentum originally the ventral mesentery unites the lesser curvature with the liver



FIG 11 Normal stomach and small intestine (a) Pylorus (b) Duodenal bulb (c) Prepyloric notch and anatomic variation (d) Feathery irregular distribution of barium in duodenum and jejunum (e) More homogeneous shadow of ileum Note difference in acuity of angle between body of stomach and pylorus in Figures 10 and 11

The greater omentum, a fatty apron, is attached to the greater curvature all along its extent except at the upper part, at which point it passes dorsally.

The stomach may be roughly described as pear-shaped or funnel-shaped with its base at the diaphragm on the left side and its apex at the pylorus inferiorly to the right. Actually, however, the stomach is extremely variable in size and shape. It not only varies with changes in the position of the individual and with the presence or absence of food, but also with the habits of the individual. Different types of stomach are described. Thus one may find (1) J type of stomach, (2) steer horn type of stomach, (3) ptotic stomach, (4) atonic stomach, or (5) hypertonic stomach. In addition to these, the stomach may change its shape and position as a result of compression or displacement by neighboring organs. A dilated transverse colon may displace the stomach markedly to the right. Enlargements of the liver, spleen, or pancreas may cause changes in the shape or position of the stomach. A kink from the esoph-

agogastric junction and the pylorus which are relatively fixed points, the remainder of the stomach is quite mobile.

The cardiac opening, which faces upward and to the right, averages 2 cm in diameter. It lies beneath the left leaf of the diaphragm, generally well below the fundus which sweeps above it. The body of the stomach is that portion between the cardiac orifice and the incisura angularis. The pyloric portion is that part below the incisura angularis. This is further divided into the pyloric antrum and the pyloric canal. The latter is surrounded by a thick muscular ring, the pyloric sphincter, which opens and closes the pyloric canal. When the pylorus is open, the index finger may readily be inserted through it.

In the average adult, gastric capacity averages 1200 cc, but this capacity varies widely.

DUODENUM

The duodenum may be described as an imperfect semicircle which is suspended at its proximal and distal ends. The first portion of the duodenum usually runs backward from the pylorus and also slightly upward and to the right. The duodenum then bends downward, forming the second portion which descends along the vertebral column. On its right, it extends to the fourth lumbar vertebra at which point it bends sharply to the left. Here the third portion of the duodenum begins and runs from right to left, usually superiorly, although this is variable. The third portion of the duodenum ends just to the left of the vertebral column, where it ascends on the spine of the upper portion of the second lumbar vertebra on the left side and becomes the fourth portion of the duodenum which bends sharply to unite with the jejunum, the sharp bend being known as the duodeno-jejunal flexure. Although the duodenum may be roughly described as ring-shaped, many variations in shape have been noted. The most common forms are the U-shaped duodenum and the V-shaped duodenum. In addition, some are ring-shaped, some C-shaped, and many are intermediate in form so that no exact description may be applied.

The length of the duodenum is quite variable and much of it is retroperitoneal. The average



FIG 12 Atonic stomach



FIG 13 Normal stomach of steer horn type in a hypersthenic individual (a) Esophageal orifice (b) Pylorus hidden behind stomach (c) Duodenal bulb hidden behind antrum of stomach

total length of the duodenum is 26 cm the first portion being 5 cm the second 8 cm the third 6 cm and the fourth 7 cm

The luminal diameter is also quite variable. The largest lumen of the duodenum is generally in the second or third portion. The fourth part is usually the smallest.

The common bile duct and the pancreatic duct open into the second portion of the duodenum approximately 10 cm from the pylorus. These ducts pass obliquely through the duodenal wall on its medial side. This point constitutes a landmark in the duodenum so that one may speak of a supraampullary, an ampullary, or an infraampullary duodenal obstruction. Each of these presents characteristic symptoms so that a specific diagnosis of localization is possible.



FIG 14 Itotic stomach with the patient in the erect position. Lateral view



FIG 16 Stomach exhibiting hypermobility with upper displacement in the prone position

The third portion of the duodenum in its passage to the left crosses the vena cava and has the pancreas above it. The head of the pancreas may overlap or encircle the second or third portions of the duodenum. This may result in duodenal obstruction particularly with persistence of the uncinate lobe. The superior mesenteric artery and vein cross over the third portion of the duodenum. Duodenal compression from this source may occur.



FIG 15 Atonic stomach in an atonic individual

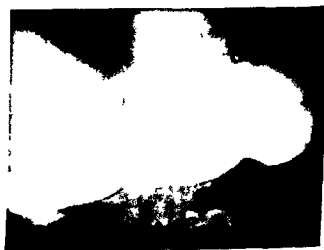


FIG 17 Transverse hypertonic type of stomach in a persthenic individual

ANATOMY OF THE GASTRO INTESTINAL TRACT



FIG. 18. Caecale stomach. Note the marked gaseous distention of the pyloric flexure.



FIG. 20. Reversed duodenal loop.



FIG. 19. Atonic stomach in the erect position filled with barium.

In an occasional patient the direction of first limb of the duodenum may be found to be reversed. It may pass downward then form a loop and sweep upward to meet the second part of the duodenum where it again passes downward. In rare instances the duodenum is found free, suspended by a common mesentery with the small bowel and colon.

LIGAMENT OF TREITZ

The very top of the duodenojejunal flexure is suspended by the ligament of Treitz. Anatomists describe the ligament of Treitz as the suspensory muscle of the duodenum. It is a bundle of involuntary muscle fibers running from the left crus of the diaphragm to the duodenojejunal angle. Cunningham describes the ligament of Treitz as a muscular band that springs from the right crus of the diaphragm on both sides of the esophageal opening. It then descends over the left crus beneath the celiac plexus and splenic and left renal vessels and the pancreas and then inserts into the duodenojejunal flexure. In children this muscle is well marked and easily isolated and some of its fibers can be traced into the root of the mesentery.

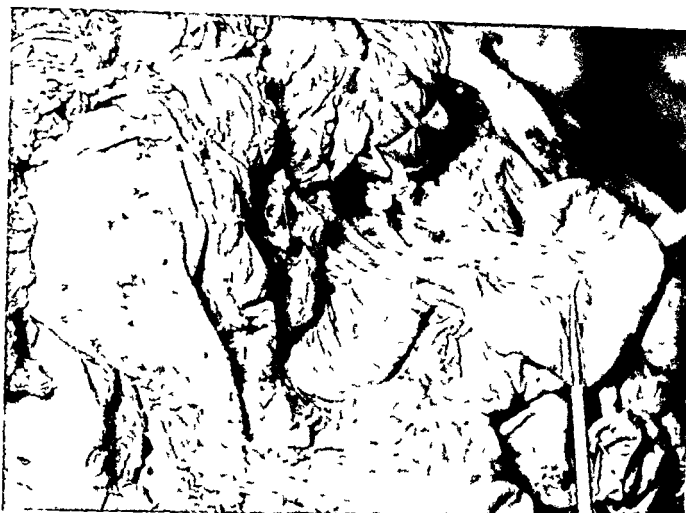


FIG. 21 Short ligament of Treitz. Note the acute angle at the duodenojejunal flexure.

where they are inserted into the peritoneum. It becomes ligamentous in the adult. It is then loose and difficult to distinguish from the surrounding fibrous tissue.

The ligament of Treitz has as its function the suspension of the duodenojejunal flexure. It tends to keep the fourth part of the duodenum at a relatively fixed point.

Anatomists have described the variation in the curve of the duodenum as a result of the variation in the position of the third portion of the duodenum. This is described as being nearly horizontal with the fourth portion nearly vertical. At times the third portion may incline upward as it passes toward the left so that it lies in line with the fourth portion.

A dissection of this area in 35 cadavers revealed considerable variation in the obliquity with which the third portion of the duodenum passed from right to left. There was a concomitant varia-

tion in the length of the ligament of Treitz associated with this. In those cadavers in which the ligament of Treitz was short the third portion of the duodenum was invariably found to angle upward at an acute angle in its passage from right to left. The shortness of the ligament of Treitz in this type of individual would result in a marked angulation at the duodenojejunal flexure. In those cadavers in which the ligament of Treitz was found to be long the third portion of the duodenum was almost horizontal in its course from right to left. In such cases the duodenojejunal angle was far less acute. In some of these cadavers the direction of the third portion of the duodenum was such that almost no angle was found between the fourth portion of the duodenum and the jejunum.

In measuring the ligament of Treitz in these cadavers it was found that the shortest ligament was 1.2 cm. long, whereas the longest was 6.25

ANATOMY OF THE GASTROINTESTINAL TRACT



FIG. 22 Long ligament of Treitz. Note that the angle of the duodenojejunal flexure is obtuse and that the jejunum is freely from this point.

These measurements were made from the per surface of the duodenum to the point of attachment of the ligament at the vertebral column. None of the cadavers (26 per cent) were found to have a ligament of Treitz that measured 12 cm in length. Each of these presented a third portion of the duodenum with a very acute angle upward and to the left and with a very acute angle at the duodenojejunal flexure. This acute angle could obstruct and in addition might become a barrier to the passage of the long intestinal decompression tube. In four of the cadavers (11 per cent) the ligament of Treitz was found to be 62.5 cm long. Associated with this long ligament the third portion of the duodenum passed almost horizontally from right to left. The duodenojejunal angle in these cases was obtuse. The long intestinal tube can readily pass an angle of this type. In 22 of the cadavers (62 per cent) the third portion of the duodenum occupied an intermediary position. In

these the ligament of Treitz measured from 18 to 37 cm in length.

When we consider that in late intestinal distention the elements present within the bowel are fluid and gas, it should be realized that the fluid heavily weights the proximal jejunal loop. The downward drag of a heavily weighted jejunum could so increase the obliquity at the duodenojejunal flexure in those cases with a short ligament of Treitz that obstruction might occur. By this same mechanism the downward progress of a long intestinal decompression tube would become difficult.

DUODENAL FOSSAE

There are five duodenal and paraduodenal fossae described by anatomists. These may on occasion cause intestinal obstruction as a result of the small intestine herniating into an abnormally large recess.



FIG. 23 Intermediary type of ligament of Treitz. Note the angle at the duodenojejunal flexure is approximately 90°.

1 The left paraduodenal fossa which lies to the left of the duodenojejunal angle opens to the right and upwards. It is said to occur in approximately one person out of five. Its borders are the aorta on the right and kidney on the left, the pancreas above and the renal vessels and the inferior mesenteric vein on the anterior wall. Hernias into this fossa are apt to cause rectal bleeding as the result of engorgement of the inferior hemorrhoidal veins.

2 The superior duodenojejunal fossa faces downward and lies in front of the second lumbar vertebra.

3 The inferior duodenojejunal fossa is directed upward and may be found in front of the third lumbar vertebra. The inferior mesenteric vein passes along its left extremity.

4 The inferior duodenal fossa is not seen very often. It may be found behind the third portion of the duodenum.

5 The mesentericoquistal fossa is found be-

hind the first part of the mesentery of the jejunum. It lies below the duodenum immediately behind the superior mesenteric artery. The fossa rarely causes bowel obstruction because it is so large that bowel may enter or leave it without becoming incarcerated. In the event that it must be enlarged, the dissection must be carried out downward away from the superior mesenteric artery in front of it.

SMALL INTESTINE

The small intestine is essentially a nonrigid mobile tube whose loops lie in every conceivable plane. It is suspended from the posterior abdominal wall by 15 cm. of mesentery. This is attached in an oblique fashion from the area to the left of the second lumbar vertebra to the right and downward to the right sacrospinous joint. The degree of obliquity of the mesenteric attachment is variable. It depends to some extent upon the line of fixation of the duodenum. Between the leaves of the



FIG. 24 Note the acute angle at the duoden jejunal flexure as shown by the kinking in the Cantor tube. Note also the downward drag of the mercury bearing balloon at the tip of this tube.



FIG. 25 The arc at the angle between the second and third portions of the duodenum is relatively gradual. There is an acute angle at the duodenjejunal flexure; a kink in the intestinal tube has developed at this point. There is marked small bowel distention.

mesentery, the arteries, veins, and lymphatics are carried to and from the small bowel. A variable amount of fat, which tends to increase appreciably with the weight of the individual, is also found between the leaves of the mesentery. The free border of the mesentery, which is attached to the small intestine, becomes very much folded. This is the natural result of the remarkable increase in length of the small bowel.

The length of the small bowel is variable. It has been reported by anatomists as ranging from 4.5 to 9.7 meters. The diameter of the small bowel, which averages 2.5 to 3 cm., shows a gradual but progressive decrease in size as it proceeds to the ileocecal valve. As a result, a mean diameter of 2 cm. is usual in the terminal ileum.

The proximal two fifths of the small intestine is called the jejunum and the distal three fifths is the ileum. It is generally quite simple to determine whether one is examining the jejunum or the ileum because of the difference in vascular pattern

in these two areas. In general, the vascular supply to the jejunum is simpler than that of the ileum. In the jejunum, the mesentery is found to be quite vascular. The vessels are long and straight, and primary arcades are generally found. The lunettes (spaces between the vessels) are extensive and translucent. In the ileum, the vascularity appears to be decreased, and the arcades become complex. No lunettes are noted. The mesentery is more or less filled with fat so that the architecture of the vessels is considerably hidden.

The uppermost third of the small bowel is usually found in the left upper quadrant. The middle third usually occupies the middle part of the abdomen and the iliac fossa. The distal third is usually found in the pelvis and right iliac fossa. There may be considerable variation in this distribution in some instances.



FIG. 23 Intermediary type of ligament of Treitz. Note the angle at the duodenojejunal flexure is approximately 90°.

1 The left paraduodenal fossa which lies to the left of the duodenojejunal angle opens to the right and upwards. It is said to occur in approximately one person out of five. Its borders are the aorta on the right and kidney on the left; the pancreas above and the renal vessels and the inferior mesenteric vein on the anterior wall. Hernias into this fossa are apt to cause rectal bleeding as the result of engorgement of the inferior hemorrhoidal veins.

2 The superior duodenojejunal fossa faces downward and lies in front of the second lumbar vertebra.

3 The inferior duodenojejunal fossa is directed upward and may be found in front of the third lumbar vertebra. The inferior mesenteric vein passes along its left extremity.

4 The inferior duodenal fossa is not seen very often. It may be found behind the third portion of the duodenum.

5 The mesentericoparietal fossa is found be-

hind the first part of the mesentery of the jejunum. It lies below the duodenum immediately behind the superior mesenteric artery. This fossa rarely causes bowel obstruction because it is so large that bowel may enter or leave it without becoming incarcerated. In the event that it must be enlarged the dissection must be carried out downward away from the superior mesenteric artery in front of it.

SMALL INTESTINE

The small intestine is essentially a nonrigid mobile tube whose loops lie in every conceivable plane. It is suspended from the posterior abdominal wall by 15 cm. of mesentery. This is attached in an oblique fashion from the area to the left of the second lumbar vertebra to the right and downward to the right sacrospinous joint. The degree of obliquity of the mesenteric attachment is variable. It depends to some extent upon the line of fixation of the duodenum. Between the leaves of the



FIG. 24 Note the acute angle at the duodenojejunal flexure as shown by the kinking in the Cantor tube. Note also the downward drag of the mercury bearing balloon at the tip of this tube.



FIG. 25 The arc at the angle between the second and third portions of the duodenum is relatively gradual. There is an acute angle at the duodenojejunal flexure; a kink in the intestinal tube has developed at this point. There is marked small bowel distention.

mesentery, the arteries, veins, and lymphatics are carried to and from the small bowel. A variable amount of fat, which tends to increase appreciably with the weight of the individual, is also found between the leaves of the mesentery. The free border of the mesentery, which is attached to the small intestine, becomes very much folded. This is the natural result of the remarkable increase in length of the small bowel.

The length of the small bowel is variable. It has been reported by anatomists as ranging from 4.5 to 9.7 meters. The diameter of the small bowel, which averages 2.5 to 3 cm., shows a gradual but progressive decrease in size as it proceeds to the ileocecal valve. As a result, a mean diameter of 2 cm. is usual in the terminal ileum.

The proximal two fifths of the small intestine is called the jejunum and the distal three fifths is the ileum. It is generally quite simple to determine whether one is examining the jejunum or the ileum because of the difference in vascular pattern

in these two areas. In general, the vascular supply to the jejunum is simpler than that of the ileum. In the jejunum, the mesentery is found to be quite vascular. The vessels are long and straight, and primary arcades are generally found. The lunettes (spaces between the vessels) are extensive and translucent. In the ileum, the vascularity appears to be decreased and the arcades become complex. No lunettes are noted. The mesentery is more or less filled with fat so that the architecture of the vessels is considerably hidden.

The uppermost third of the small bowel is usually found in the left upper quadrant. The middle third usually occupies the middle part of the abdomen and the iliac fossa. The distal third is usually found in the pelvis and right iliac fossa. There may be considerable variation in this distribution in some instances.

The vascular patterns of the small bowel have been the subject of a number of investigations. Formerly it was believed that the collateral circulation stopped with the terminal row of arcades in the mesentery of the small bowel. Beyond this point there was supposedly no anastomosis either between the *vasa recta* in the mesentery or between the ramifying vessels on the bowel wall. Noer studied the blood supply of the jejunum and ileum in a comparative study of man and certain laboratory animals. He found that the human mesenteric vascular pattern consisted of multiple mesenteric arcades which increased in number and complexity as one passed from the jejunum to the ileum. The *vasa recta* proceeded from these arcades to the intestinal wall without any intercommunication. They then coursed to either side or to both sides of the intestines as mural trunks. Up to this point the observations of Noer were similar to those of Collins. From this point on Noer demonstrated that a free system of anastomoses appeared in the intestinal wall and that three distinct types of anastomosis could be recognized: (1) direct communications between the mural trunks of both sides; (2) a plexiform type of anastomosis; and (3) transverse vessels joining arcuate mural anastomoses. From these studies a very rich collateral blood supply was noted in the bowel wall of humans. In a later study of the vascular patterns in man, Ross noted that the arteries followed a definite pattern. The mesenteric vessels formed several arcades in the jejunum. These increased in number and complexity as one passed from the jejunum to the ileum. In the jejunum anastomoses between the roots of the *vasa recta* were infrequent and the vessels ran without any union with their neighbors straight to the bowel wall. In the bowel wall they gave off a series of short vessels and branched repeatedly forming free communications with their neighbors. In this fashion the *vasa recta* formed complete arterial circles around the bowel. In some instances there was a junction with the vessels from the opposite side across the antimesenteric border. By means of this rich arterial pattern a free flow of arterial blood is assured to the bowel wall at all times. In addition to this rich vascular bed free anastomoses between the *vasa recta* on the wall of the small in-

testine was formed by fine vessels branching from them to meet their neighbors on either side forming longitudinal anastomatic channels. Because of these channels moderate degrees of intestinal distention would not seriously impair the blood supply to the small bowel. In addition the small bowel can tolerate a considerable sacrifice of its blood supply from mesenteric injuries because of this rich vascularity. It has been demonstrated experimentally that 8 to 10 cm. of mesentery may be separated from the bowel with complete recovery because of this rich intercommunicating vascularity.

Wilms noted in 1906 that all broad flat shaped structures within the abdomen such as the mesentery may present defects or rents which appear to be congenital in origin. This is supported by the absence of inflammatory reaction circumjacent to them. The importance of these openings resides in the possibility of herniation of small bowel through them with resultant incarceration or possible strangulation of the bowel. In the fetus it may often be observed that the iliocecal branch of the superior mesenteric artery circumscribes by an anastomosis with the first of the intestinal arteries an area in the mesentery of a well rounded or oval shape. This area is remarkable in that it prevents no fat, no visible blood vessels of any kind and is never occupied by any mesenteric glands. While this clear area is not constant it is sufficiently frequent to be common knowledge. It is in this identical area that the mesenteric rent or hole is most commonly found. In an experimental case the mesentery in this area is thin, cribriform and punctured by many small holes. Treves reports that this area is by far the most common site for such defects.

II ILEOCECAL VALVE

Colonic function normally begins with the entrance of intestinal contents into the cecum through the ileocecal valve. This valve permits the passage of intestinal contents into the colon while preventing its reflux. The ileum terminates in the cecum at an oblique angle and invaginates the cecal wall. As a result of this obliquity a tendency toward a valve like action results. However, the invaginated wall forms two transverse lips about

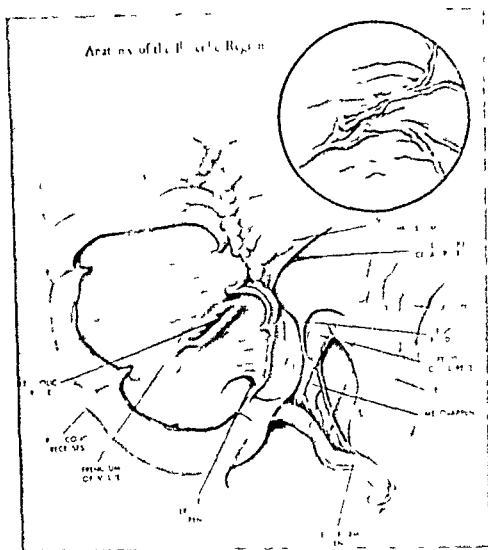


FIG 76 Normal anatomy of the ileocecal region Notice the liplike ileocecal orifice

the ileocecal valve. One of these is above and the other below the orifice. The lips unite laterally to form a fold on each side of the opening and continue around the interior of the bowel. Distention of the cecum causes stretching of these lips. This draws them together, thus closing the ileocecal valve. The arrangement is such that the opening when closed can withstand considerable pressure from within the cecum but is readily opened from the ileal side. On the cecal side, the fluid content may accumulate even though the cecum is filled with considerable fluid because the ileum is still able to eject a fecal stream against the weight of this column of liquid. This power of the ileum is based upon the fundamental law of hydraulics

which states: if pressure is exerted through a narrow water-filled tube with a cross section of 1 square centimeter upon a water-filled chamber with a cross section of 1 square meter, each square centimeter of the larger chamber will exert a pressure equal to that exerted by the water in the small tube. As a result, the force at the surface of the water in the larger chamber is increased 10,000 times.

Variations in the structure and competence of the ileocecal valve have been found to be associated with variations in the development of the cecum. The cecum is slow to assume its adult form in humans. Because of this, large numbers of imperfectly developed ceca can be found in hu-

GASTRO INTESTINAL OBSTRUCTION

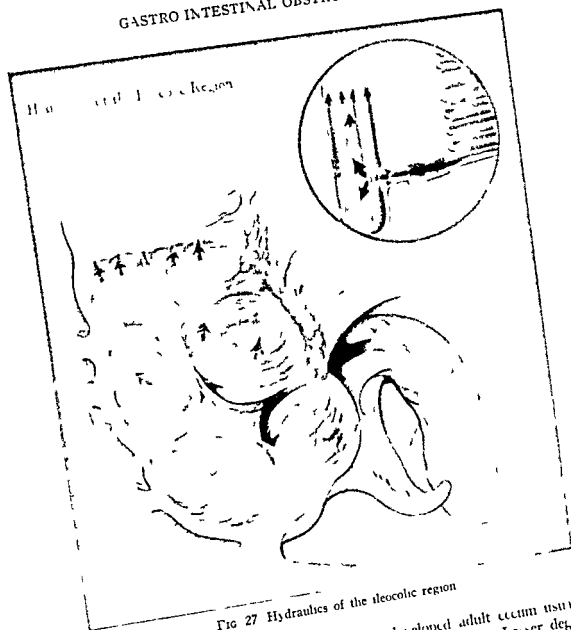


FIG 27 Hydraulics of the ileocecal region

mans. With this there is a concomitant variation in competency of the valve. In the course of its development the posterior valve frenulum appears first being followed by the anterior at a later time. These are formed by a transverse fold in the wall of the cecum at the point at which it joins the ileum. This fold is represented on the external surface of the cecum by a crease which extends transversely from the ileocecal junction to beyond the anterior and posterior tenia. These do not dip into this crease but span the gap. By so doing considerable support is given to the frenulum of the ileocecal valve. The medial tenia is particularly important in strengthening the valve very close to the orifice. If this middle tenia is torn a competent ileocecal valve becomes incompetent.

A fully developed adult cecum usually has a competent ileocecal valve. Lesser degrees of competency are associated with incompletely developed cecum. Contrary to popular belief perfectly functioning competent valves are in the minority. In a comprehensive review of the competency of the ileocecal valve is shown by reflux of barium into the ileum during the course of barium enema study radiologists reported that 65 to 90 per cent of the valves studied were incompetent. This would suggest that the majority of ceca are not perfectly developed in man.

There are various theories concerning the mechanism by means of which the valve function. The two most commonly held views are that the ileum is separated from the cecum by a true valve or

that the ileum is separated from the cecum by a sphincter muscle

The colonic contents are propelled along the colon from the cecum by mass movements. This represents a mass peristaltic activity which consists of infrequent contractions which sweep the contents along, en masse for considerable distance. These movements normally occur as infrequently as two to three times a day. They may be associated with the ingestion of food by means of the gastrocolonic reflex. In certain individuals these movements may also be initiated by psychic factors such as the sight, smell or thought of food.

THE LARGE INTESTINE

The large intestine from the cecum to the rectum varies from 1 to 1.9 meters in length. Its luminal capacity decreases from the cecum as it approaches the rectum where it again dilates. Its luminal diameter is quite variable depending upon its state of distention. It is subdivided into the cecum, the ascending colon, the transverse colon, the descending colon, and the sigmoid colon, which is composed of the iliac colon and the pelvic colon.

Cecum

Under normal conditions the cecum is found in the right iliac fossa. However, because descent and fixation of the cecum occurs relatively late in the developmental process, variations in position of the cecum are common. It may be found in a subhepatic position or may be found to overlie the brim of the pelvis. Its degree of fixation is also variable from complete fixation in an almost retroperitoneal position to nonfixation with a mesentery and a completely encircling peritoneal coat. The cecum is the first portion of the colon and it ends as a blind pouch. The ileum normally opens into its medial side and the appendix also opens into it. The average length of the cecum is 6.25 cm and it is capable of tremendous distention. The tenia coli continue on the cecum and terminate at the origin of the appendix. One band is placed anteriorly and the other two are placed medially and laterally, respectively.

Ascending Colon

The ascending colon continues from the cecum in an upward and backward direction until it reaches the under surface of the right lobe of the liver. At this point it bends sharply to the left forming the hepatic flexure. It lies in the right flank and rests upon the psoas and quadratus lumborum muscles. It also lies in front of the lower end of the right kidney. The mesenteric attachment of the ascending colon is variable. It may be entirely retroperitoneal or may be completely covered by peritoneum and have a mobile mesentery. In cases of this type the stage is set for the possible development of cecal volvulus.

Transverse Colon

The transverse colon runs obliquely from right to left and upward from the hepatic flexure to the splenic flexure. As it crosses the abdomen it sags more or less in the middle. This may be so pronounced as to bring it to the pelvic brim. It lies on the anterior surface of the right kidney and over the third portion of the duodenum. The splenic flexure is much more acute and at a considerably higher point than the hepatic flexure. The splenic flexure is held in position by its attachments to the spleen, pancreas, and diaphragm. The transverse colon is covered anteriorly by the greater omentum and is attached to the greater curvature of the stomach by the gastrocolic ligament. Rents and holes in the greater omentum of congenital or man-made origin are not infrequent sources of herniation of bowel and strangulation.

Descending Colon

The descending colon begins at the splenic flexure and passes downward in the left flank. It passes into the sigmoid colon at the crest of the ileum. It lies anterior to the left kidney and is covered with peritoneum anteriorly and on its sides. Its posterior surface is bare of peritoneum. At times the peritoneal covering is complete.

Sigmoid Colon

The sigmoid colon is described as consisting of two distinct parts: (1) the iliac colon which has no mesentery and ends at the medial border of

the psoas where it becomes the pelvic colon, and (2) the pelvic colon which begins as noted and opens into the rectum. The pelvic colon is suspended from the posterior abdominal wall by a definite mesentery which permits considerable mobility. There are three recognizable types of pelvic colon. The most common type lies in contact with the left pelvic wall, then passes across to the right side of the pelvis and bends backward towards the midline where it turns down to join the rectum at the third portion of the sacrum. The second type of pelvic colon appears longer. It passes over to the left pelvic wall making an S curve before joining the rectum at the usual level. In the third type of colon the bowel is abnormally short and may be devoid of mesentery. Normally the mesentery of the pelvic colon is fan shaped with the narrow portion of the fan attached to the posterior abdominal wall. This gives the pelvic colon a narrow pedicle which may be further narrowed by contraction secondary to inflammatory processes, endometriosis or neoplastic infiltration. This portion of the gastro intestinal tract has as its sole function the storage of fecal material. It has little or no absorptive function and it is capable of secreting only mucus.

Excessive lengths of pelvic colon may be classified into three groups. In the first group are found those cases in which there is a persistence of a fetal type. This would be the result of an arrest in development and is considered as being a hereditary trait. The colon may lie in the right abdomen and take the form of an inverted U. In such cases since the limbs of the U are closely approximated, an axis of rotation is present which predisposes to volvulus. In the second type the megasigmoid is associated with gross dilatation of the lumen which may occur alone or in conjunction with a general colonic enlargement. It does not in itself predispose to volvulus. The third, the acquired type of elongated colon, is the result of adaptation to dietetic habits in which coarse foods are eaten. This is rather common in Russia and the Baltic area and is a frequent cause of volvulus.

Blood Supply to Colon

The right colon is supplied by the superior mesenteric artery and its branches. These are the mid-

dle colic, the right colic, and the ileocolic. The superior mesenteric artery arises from the aorta, passes behind the pancreas, crosses the third portion of the duodenum and then enters the base of the mesentery. The superior mesenteric vein returns blood from the small bowel, the ascending colon and right half of the transverse colon. This vein unites with the splenic vein behind the neck of the pancreas to form the portal vein which passes upward into the liver. The left colon is supplied by the inferior mesenteric artery which arises from the aorta 2 to 6 cm. from the bifurcation of the aorta. Measuring from the promontory of the sacrum, the origin of the inferior mesenteric artery is found to be from 7.5 to 13.1 cm. As it passes downward from its point of origin it gives off three arteries which supply the left colon and upper rectum. These are the left colic, the sigmoidal and the superior hemorrhoidal arteries. The inferior mesenteric vein is an upward continuation of the superior hemorrhoidal vein to which is added the sigmoidal and left colic veins. The inferior mesenteric vein passes upward lateral to the artery. It then curves to the right above the duodenojejunal flexure and empties into the splenic vein. Compression of this vein by *paraduodenal fossa hernias* is not uncommon. Rectal bleeding then follows.

In contrast to the small bowel, the colon is comparatively avascular. The free communication between the vasa recta noted in the small bowel is not nearly as free in the colon. An imperfect arcade system is developed in places along the colonic wall. It cannot compare in abundance with the connections of the blood vessels noted in the small intestine. Although there are some anastomoses across the antimesocolic border between the long vessels of opposite sides, these are infrequent. As a result, it should be obvious that the colon cannot stand as much distention as the small bowel. The demonstration of vascular loops in the bases of the epiploic appendices is still controversial.

RECTUM AND ANUS

The rectum begins as a termination of the pelvic colon in front of the third sacral vertebra. It has no mesentery, tenui coli or epiploic appendices. Its upper third is covered anteriorly and at the

sides by peritoneum while its posterior surface is bare. Its middle third is covered only anteriorly by peritoneum and its lowest third is entirely devoid of peritoneum. The rectum is approximately 11 cm. long. It follows the curve of the sacrum and coccyx and ends in front of the coccygeal tip. At this point it bends sharply downward and backward into the anal canal. This canal is the terminal end of the gastro-intestinal tract. It is approximately 3.5 cm. long. It runs downward and backward from the rectum. It passes through the fascia between the levator ani muscles and out onto the perineum.

The rectum is supplied arterially by five arteries. In descending order these are the superior hemorrhoidal from the inferior mesenteric, two middle hemorrhoidals from the internal iliac and two inferior hemorrhoidals from the internal pudendal. Analogous veins return venous blood along similarly named vessels.

ILEOCECAL AND SIGMOID FOSSAE

The superior and inferior ileocecal fossae are found in the ileocecal region. These are pouches variable in occurrence, size and depth. The importance of the cecal fossae in connection with bowel obstruction lies in the remote possibility that the bowel may herniate into an abnormally large one.

The intersigmoid fossa is a small peritoneal pouch present in approximately 75 per cent of individuals. It is found on the under side of the mesosigmoid flexure and is a result of the failure of the mesosigmoid to unite with the peritoneum of the posterior abdominal wall. Its orifice is generally circular and ranges in diameter from 1 to 3 cm. Herniation of the small bowel or colon through this opening is possible. In such cases strangulation occurs early.

VEILS, BANDS, ADHESIONS

The occurrence of structures described as bands, veils, adhesions or ligaments in the right upper quadrant has been known for 90 years. Hirsch described the hepatoduodenal ligament in 1845. Since then there have been numerous anatomic studies regarding these interesting structures. An

atomically, duodenal bands are believed to represent the unattached portion of the free edge of the lesser omentum. Depending upon the course of these anomalous membranes they are called hepatoduodenal, hepatocolic or cystocolic ligaments. They either cross or attach to the duodenum in its first or second portions. When the first limb of the duodenum is involved radiologically demonstrable duodenal deformity occurs. Bands involving the second portion of the duodenum may cause a characteristic radiologic appearance. Examination of the patient in the erect A-P position immediately after the stomach has begun to empty causes the second limb of the duodenum to be pulled to the right instead of distending in its normal fashion. In some cases a sharp angle may result between the fixed upper and the free lower portion which acts as a point of irritation causing temporary duodenal obstruction. Most of the bands in the right upper quadrant are of congenital origin. Acquired adhesions due to infection, trauma or previous surgery may also occur.

PERITONEAL FAT LINE

The peritoneal lining of the abdominal cavity consists of a thin wall of endothelial cells. Next to this layer is a layer of fat containing some connective tissue. This fat lies between the peritoneum and muscle. It varies in thickness in different individuals. It can be seen on a survey film of the abdomen as a dark stripe in the flanks next to the bowel. With an accumulation of fluid in the abdomen or edema of the peritoneal endothelial cells an edema of this fat layer occurs. This brings the absorptive capacity of roentgen rays of the fat layer up to the neighboring muscle and the contrasting dark stripe is then lost. This results in a homogeneous shadow in the flank. When this fat line is absent in a well taken film that is not overexposed it is an indication of fluid in the abdomen. The fat line is of greatest diagnostic significance when it is absent in only one part of the flank. If associated with a local paralytic ileus this is indicative of a localized abscess or localized peritonitis.

PHYSIOLOGY OF THE GASTRO- INTESTINAL TRACT

An understanding of normal physiology is essential to the proper understanding of physiologic derangements causing obstruction. This is particularly important in obstructive lesions of the cardioesophageal junction. A knowledge of physiology is essential both to best utilize its mechanisms in intestinal intubation and to utilize the effect of drugs upon intestinal motor activity. There are in addition varieties of intestinal obstruction based solely upon deranged physiology.

MECHANISM OF DEGLUTITION

The esophagus is a musculomembranous tube whose sole function is the transport of food from the mouth to the stomach. The mechanism by means of which this is accomplished may be divided into three phases.

1 The buccopharyngeal phase begins with the oral nasal and laryngeal orifices closed off by the lips, tongue and soft palate. The mylohyoid muscles contract thrusting the bolus downward. At this time relatively high pressures develop in the oropharynx and a single positive wave is developed. These events occur in less than one second.

2 The esophageal phase follows the buccopharyngeal phase. This begins with a relaxation of the cricopharyngeus muscle. At this time the entire esophagus is said to relax. Liquids drop immediately to the ampullary portion of the esophagus as a result of gravity. Solid material passes by slow peristaltic movements down the esophagus.

3 The cardioesophageal phase begins when the swallowed material reaches the ampulla of the

esophagus. It pauses at this point. The distal end of the esophagus at the stomach relaxes periodically permitting gushes of food to enter the stomach.

It has been amply demonstrated that liquids drop down the esophagus regardless of the slow peristaltic waves. The peristaltic waves appear to function autonomously. They are initiated voluntarily by the pharynx and are completed by the intrinsic myenteric reflex. Balloon experiments, however, have demonstrated peristaltic waves independent of pharyngeal activity. The rhythm of esophageal peristalsis is not comparable with that of the small bowel.

Two distinct peristaltic wave types are described as occurring in the human esophagus. The primary peristaltic wave travels to the distal esophagus and is the main propelling force for food. A wave of relaxation precedes the wave of contraction. The secondary peristaltic wave depends upon intraesophageal distention for its stimulation. It generally begins in the upper third of the esophagus.

In addition to its peristaltic activity the esophagus shows segmental activity. This manifests itself as spasm and in itself is not pathologic.

Abnormalities in Mechanism

Antiperistalsis may be demonstrated at times independent of esophageal motility in the presence of lower esophagus obstruction of long standing like that occurring with stricture. Neurologic disease may result in paralysis of the esophagus in its proximal third. This may be due to bulbar poliomyelitis, diphtheria or myasthenia gravis.

Once the food reaches the lower two thirds it progresses normally because of the intrinsic automatic tonus of this portion of the esophagus. Tumors of the cerebellopontine angle involving the acoustic nerve often disturb the nerves involved in deglutition. Dysphagia then results

Effect of Esophageal Lesions

The esophagus reacts to injury or inflammatory processes by spasm. This is a persistent accentuation of a normal physiologic process.

Effect of the Diaphragm

The role of diaphragmatic activity upon esophageal emptying is controversial. Although there seems to be some correlation between the normal esophageal peristaltic activity and the pharyngeal mechanism at the cardia of the stomach, the effect of the diaphragmatic action has not been settled. Palmer sums up the available evidence in concluding that the diaphragm has little influence over the esophageal function.

Psychosomatic Aspects

Under normal conditions there is no voluntary control over the muscular activity of the esophagus except for the initiation of the process of deglutition. It has been clinically demonstrated, however, that some individuals can be taught to relax the esophagus to such a degree that radiographic demonstration is possible. The exact role that psychosomatic disturbances play in the development of cardiospasm has not been entirely clarified. On the basis of psychiatric study of such patients the suggestion has been made by some gastro-enterologists that cardiospasm represents a form of conversion hysteria. The term psychosomatic has been applied to this disorder suggesting that it originates in the sort of high strung individual with whom we commonly associate peptic ulcer, hypertension and coronary disease.

STOMACH RESERVOIR AND SPHINCTERS

The stomach is a muscular reservoir in which food is held after ingestion. Here it is prepared for further digestion and is released in small amounts. This mechanism is a very complex one

and therefore considerable variation is possible. The sphincters as well as the stomach itself are not only sensitive to psychogenic stimuli but are also sensitive to stimuli from the senses as well. For this reason disturbances in the physiology of this organ are quite common.

Cardiac Sphincter

The cardiac sphincter is commonly considered to be the muscular ring around the terminal end of the esophagus. There appears to be considerable variation of opinion, however, as to the presence of this sphincter. The dissenters to this concept point out that in man the thickness of this muscle is no greater than that of the rest of the tube. Animal experiments have demonstrated a vagal as well as a sympathetic innervation. The exact mode of action of each, however, is somewhat confusing. Stimulation of the vagus in experimental animals has resulted at times in contraction of this area and at times in relaxation. It seems that the character of the response depends upon the degree of tonus of the sphincter at the time of stimulation. Observations in the clinical treatment of cardiospasm in humans are suggestive of the inhibitory effect of the vagus. No definite statement is possible as to the exact effect of the sympathetics upon the sphincter. Both stimulating and relaxing effects have been ascribed to its action. Under normal conditions the cardiac sphincter relaxes to permit the passage of the oncoming bolus of food propelled downward by the peristaltic contraction wave of the esophagus behind it. There is thus a well timed well integrated mechanism permitting the discharge of small amounts of food from the lower esophagus into the stomach.

Abnormal Function. Derangements in the normal physiologic mechanism at the cardiac sphincter are quite common. They can be induced by a wide variety of stimuli. The effect of emotions such as fear, shame or sorrow are well known. The tonus may be increased abnormally by a strong stimulus applied to the mouth, pharynx or nose. Trauma to the nasal mucosa caused by inept attempts at passage of a long intestinal tube often result in cardiospasm which precludes passage of the tube into the stomach.

Strong stimuli applied to the stomach may also result in reflex increased tonus in this area

Pathologic Physiology Persistent spasm of the lower esophagus and cardiac sphincter is not infrequently seen in psychoneurotic individuals. Although the muscular contraction of the lower esophagus which occurs with nervous states is usually transitory, a radiologically demonstrable degree of severe spasm may occasionally occur. Achalasia of the esophagus called cardiospasm by some may produce a complete functional obstruction of the lower esophagus. No organic basis for this type of obstruction has been proved. In advanced achalasia the esophagus dilates to a degree rarely seen in mechanical obstructions of an organic nature. The consensus at present is that cardiospasm results from a neuromuscular dysfunction of the lower esophagus proper. The myenteric ganglion appear to be involved but the cause is unknown. It has been suggested that in this latter respect it bears a similarity to Hirschsprung's disease.

Gastric Reservoir

Gastric tonus is subject to wide physiologic variations. Emotional reactions, sensory stimuli, fatigue, and the type of food ingested directly affect it. In general, pleasant stimuli increase tonus whereas unpleasant or painful ones depress it. Electrolyte deficiencies due to loss of potassium and chlorides result in general loss of muscular tonus. Long standing debilitation due to infections and starvation or vitamin deficiencies results in a loss of tonus. Gastric atony may occur on occasion for no ascertainable reason. Swallowed food drops into the stomach and promptly falls to the bottom. Hypertonus may similarly occur in tense nervous individuals and may be a manifestation of a psychotensive state.

Peristaltic activity of the stomach is also subject to wide physiologic variation. Anxiety, hostility, or resentment increases the peristaltic activity whereas sorrow and sadness depress it. Normally ingested food remains in the stomach for several hours. During this period of time the food is trapped by the closure of the cardiac and pyloric sphincters. The fundic portion of the stomach normally serves as a reservoir for retaining

the bulk of the food while the muscular pyloric portion (antrum) is the apparatus which mixes it and forces it through the pylorus. It has been demonstrated radiologically that shortly after the entrance of food into the stomach small contractions start in its middle and run toward the pylorus. These waves appear at regular intervals and become more and more forceable, pressing the gastric contents against the pylorus. The pyloric ring relaxes at intervals and the antral contractions force some of the liquified food through it. In addition the stomach reacts to differences in temperature of its ingesta. Ice water invariably initiates vigorous peristaltic waves. This has long been known and utilized by radiologists. A specific reaction to different types of food also occurs. Carbohydrate foods begin to pass out of the stomach soon after ingestion and require only half as much time as proteins. Fats when taken alone remain within the stomach for the longest time. Hydrochloric acid in the stomach seems to favor or produce relaxation of the pyloric ring according to Cannon although it causes a contraction of the duodenum.

The influence of hot and cold applications upon the gastric motor activity has been the subject of considerable experimental study. Although most radiologists appear to be agreed that the ingestion of cold increases peristaltic activity,illard and Nye, Carlson and Jaworski reported that iced water taken by mouth diminished gastric tonus and peristaltic contractions. Todd in his studies of gastric motility found that both hot and cold drinks of milk increased the frequency and vigor of peristaltic waves. The stimulation appeared to be greater and more prolonged with hot drinks than with cold.

Distention of the stomach acts as a normal stimulus of peristaltic gastric activity. Overdistention however inhibits it.

Alcohol, coffee and baking soda in therapeutic doses increase peristaltic activity whereas smoking, atropine, thiamin deficiencies and severe infections depress it.

The stomach is extrinsically supplied by the right and left vagus nerves as well as by the sympathetic gastric plexus. It also contains an intrinsic plexus of Meissner and of Auerbach such as are

found throughout the gastrointestinal tract. Stimulation of these nerves results in unpredictable peristaltic activity. The effect obtained seems to depend upon the strength of the stimulus and the tonus of the stomach at the time the stimulus was applied. In general however, the vagus nerves are considered as being motor to the stomach and inhibitory to the sphincters, whereas the sympathetic nerves are inhibitory to the stomach and motor to the sphincters. The relaxed dilated stomach, so often resulting from bilateral vagotomy, is known to all surgeons.

Under normal conditions the quantity of food ingested appears to influence intragastric pressure very little. The stomach adjusts itself to varying quantities of ingested material by relaxing. As a result its capacity may be considerably increased without increasing the intragastric pressure. In addition the increase in gastric capacity is accompanied by a relaxation of the musculature of the abdominal wall. When a breakdown of this mechanism occurs after a meal eaten rapidly and under tension, then there is a feeling of pressure in the upper abdomen due to a failure of the gastric wall to relax with sufficient rapidity.

Tremendous dilatation of the stomach may occur as a result of markedly afferent stimuli like those resulting in acute gastric dilatation after upper abdominal surgery. Poliomyelitis may cause tremendous gastric dilatation as a result of damage to the anterior horn cells caused by the virus. Multiple sclerosis and paralysis agitans may result in spasm of the pylorus to a degree causing severe retention. The passage of a long intestinal decompression tube through a pylorus of this type is almost impossible.

The effect of complete loss to the body economy of the gastric secretion was well demonstrated by Dragstedt in 1930. In a rather ingenious experiment the entire stomach of a dog with its secretory innervation and blood supply intact was isolated from the continuity of the alimentary tract. As a result all the gastric secretion would permanently drain away. Food passed directly from the esophagus to the upper end of the duodenum. A stomach isolated in this fashion secreted a large amount of gastric juice, ranging from 600 to 2600 cc. in a 24 hour period. The acidity and

pepsin strength of this secretion were high. The cephalic phase of gastric secretion was not interfered with nor was the hormonal phase. As a result of the loss of gastric secretion a profound change in electrolytes occurred, a marked loss of chlorides, an increase in carbon dioxide combining power, and a marked alkalosis also occurred. The nonprotein nitrogen and urea increased. The animals experimented on recovered from the effect of the operation and seemed to be in good condition for two or three days. Lean meat and water were taken in small amounts although supplied freely. The stools were well formed. The animals lost weight steadily however. After two or three days they refused food and water and became weaker and depressed. Large amounts of highly acid gastric juice were secreted by the isolated stomach however during this period of time. We see here profound changes in chemistry due to the loss to the body of gastric secretion whose essential elements in this regard are water, sodium, and chloride.

Pyloric Sphincter

Under normal conditions the pyloric sphincter contracts rhythmically from three to five times per minute. The movements of the pyloric antrum sphincter and duodenum appear to be correlated by means of their intrinsic nerve plexuses. As a result the duodenum is relaxed when the pyloric antrum contracts. Each contraction of the pyloric sphincter occurs during the phase of duodenal relaxation. The reciprocal relationship is such that movements in the duodenum have been shown to influence the activity of the pyloric sphincter. Duodenal stimulation which initiates a peristaltic wave also results in a firm contraction of the pyloric sphincter. By this mechanism the status of the duodenum indirectly controls the emptying of the stomach. When the duodenum is emptied its contractions subside. The pylorus then relaxes thus permitting further gastric emptying. In addition to its effect upon the sphincter the status of the duodenum also has a profound effect upon the peristaltic activity of the antrum. Substances placed in the duodenum cause a marked reduction in force of antral peristaltic activity whereas emptying of the duodenum results in greatly increased

gastric motility and, as a result an increased speed of evacuation

The concept formerly held by Cannon that the pylorus was controlled by acidity of the gastric or pyloric contents has been given up. A stimulus of any type applied to the duodenum may reflexly increase the tonus of the pyloric sphincter. Hydrochloric acid can serve as such a stimulus despite the fact that the acid *per se* is not the responsible factor. This can readily be shown by the fact that a high degree of alkalinity may also delay gastric emptying. It has been clearly demonstrated that there is no relationship between gastric emptying time and gastric or duodenal acidity.

An important factor in determining the rate of gastric emptying is the degree to which the gastric contents have been reduced to a semifluid consistency. The observation of Cannon and Grutzner that material may remain in the fundic portion of the stomach for long periods of time until the gastric tonus forces it out toward the antrum still holds true. Cannon's observation that solid objects forced against the pylorus prevent relaxation of the sphincter and retard the passage of gastric chyme may explain the rapidity with which semi-liquid contents are evacuated. This important observation is utilized in the passage of the mercury-bearing balloon tipped single lumen intestinal decompression tube. This tube presents to the antrum and pylorus a sac of fluid contents simulating the semifluid gastric contents. As a result a much shorter emptying time or passage time through the pylorus occurs. Best and Taylor speak of the possible retrograde waves set up in the gastric vestibule as a result of solid particles coming into contact with the pylorus. The proper utilization of these two principles in the construction of a mercury-bearing intestinal decompression tube should not be minimized.

Pylorospasm

Persistently acting stimuli may result in a persistent spasm of the pylorus. This may be psychotensional in origin due to the stresses and strains of living and working in a highly competitive atmosphere. Disease in adjacent organs such as gall bladder disease, pancreatitis, duodenal ulcer or reflex stimuli from distant areas may result in a clinically

recognizable degree of pylorospasm. Trauma to the nasal mucosa due to inept attempts at passing an intestinal tube may result in a spasm at the pylorus which will trap the tube within the stomach. The constant irritation within the nose may so upset the patient that the normal relaxation of the pyloric sphincter is absent or at least decreased to such a degree that a tube will not pass. This is suggested by the fact that in such cases increasing the amount of mercury within the tube held to 8 or 9 cc., giving the patient an injection of morphine for its euphoric effect and atropine for its relaxing effect upon the sphincters will invariably result in a successful passage.

DUODENUM

The physiologic movements of the duodenum are very complex. The first portion of the duodenum has been regarded by some observers to be functionally as a part of the stomach. Its glands secrete an alkaline fluid and its junction with the second portion of the duodenum is described as being marked by a circular bundle of muscle fibers believed by some physiologists to have sphincteric action.

When the chyme from the stomach enters the duodenum its bulb contracts and the second and third portions of the duodenum undergo peristaltic antiperistaltic and segmental movements. The contents of the duodenum become churned to and fro before being passed on to the small bowel. This normal physiologic mechanism is very delicately balanced and is easily upset by disease either intrinsic or extrinsic to the duodenum. Hyperperistalsis and sphincter-like spasms of the circular layer of muscle may result in a writhing action. This type of dysfunction may be the result of vagal overactivity (vagotonia). On the other hand overactivity of the sympathetics (sympathetico-tonia) may result in dilatation of the duodenum with stasis.

Physiologic studies by Morton and Sullivan have demonstrated a striking difference between the secretory activity of the duodenum as compared with the ileum. In the duodenum a rapidly increasing secretion occurred as a result of distention as compared with a relatively inert secretion in a segment of ileum. This increase in fluid

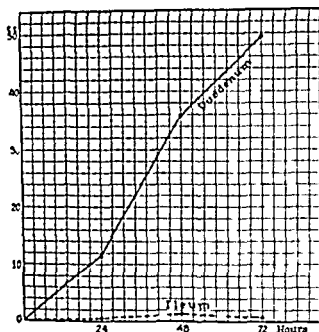


FIG. 28. A comparison of the secretion rates of equal-sized closed segments of duodenum and ileum.

content was accompanied by a fixed intra-enteric pressure which could be well demonstrated at the end of 24 hours. A rapid production of fluid accumulating in the duodenum occurred far in excess of that produced in the ileum. The secretion increased rapidly on the second day and still more rapidly by the end of the third day. As a result the pressure in an isolated duodenal loop becomes great enough to rupture it. Antonicic and Lawson demonstrated that denervation did not interfere with the building up of pressures in obstructed loops. The motility pattern of denervated loops did not change in the obstructed loops. In all cases studied the denervated obstructed bowel behaved similarly in its motility to that of the non-denervated obstructed bowel.

Effect of Total Loss of Pancreatic Juice

Elman and McCaughy demonstrated in 1927 that total loss of pancreatic juice has fatal results. Dragstedt reached the same conclusion in his experimental studies utilizing a new type of pancreatic fistula. In all animals in which there was a total loss of pancreatic juice such as occurs in high intestinal obstruction below the level of the pancreatic duct, a characteristic change occurs in

the chemistry of the blood. A decided decrease in the concentration of fixed base and to a lesser extent a decrease in chlorides result. The HCO_3 decreases markedly with a resulting shift of the pH toward the acid side. The experimental animals lose weight rapidly and become weak and depressed. Death occurs within three to six days or the animals become moribund. The most striking changes are severe tissue dehydration and acidosis. The clinical picture is one of severe depression.

Effect of Total Loss of Bile

Humans as well as experimental animals can withstand a total loss of bile for a relatively long period of time. The symptoms that result are referable to a disturbance in digestion due to the absence of bile in the intestine and to a much lesser extent to the loss of substances secreted in it. Of the substances lost bile salts are probably of the greatest significance. A complete biliary fistula with the gall bladder removed results in a greater loss of chloride than if the gall bladder is present. This is the result of a loss of the gall bladder mucosa which has the capacity to reabsorb chloride. In either event the disturbance is of a chronic nature clinically and does not present the acute catastrophic features seen when complete loss of pancreatic juice occurs.

The loss of sodium and chloride ions reduces the capacity of the body to hold water. As a result a severe degree of dehydration is produced. The sodium ion appears to exert a much greater effect in this regard than does the chloride. This was demonstrated rather conclusively by Gamble. After a total loss of gastric or pancreatic juice for four to five days the dehydration is extreme. Experimental animals under such conditions do not attempt to replace the lost water by drinking even though water is freely available. From this and other studies it has been suggested that thirst is related not so much to actual dehydration as to a disproportion between the total amount of water and the electrolytes dissolved in the body fluids. Water alone given by mouth or stomach tube will not relieve this type of dehydration. This same holds true for 5 per cent glucose solution in water given intravenously. The water is rapidly excreted

in the urine under these conditions. Giving such animals Ringer's solution of sodium and chloride rapidly results in a recovery. The animals begin to eat and drink in a normal fashion. The blood and tissue chemistry return to normal values. From this it is obvious that the essential elements removed in the pancreatic and gastric juices are the sodium and chloride elements.

SMALL INTESTINE

Certain facts are generally accepted with regard to the physiology of the small intestine yet much remains to be learned. The propulsive movements of the small bowel are composed of contractions of both the circular and the longitudinal muscle layers. These are preceded and followed by relaxation. The intestinal peristaltic movements are regarded as reflexes which are coordinated and controlled through the intramural myenteric plexus. To be effective and normal such movements require the proper function of the nervous apparatus in the bowel wall. It has been repeatedly demonstrated that the first evidence of fatigue of the bowel musculature is its failure to relax in front of a contraction wave. As a result of this there is a dissociation of the intestinal peristaltic movements resulting in a failure of normal propulsion. The mucosa is said to move independently of the muscle layer by virtue of its muscularis mucosa. The latter forms and changes the mucosal folds resulting in the characteristic feathery pattern of the small bowel noted radiologically.

The small intestine is innervated by the vagus (parasympathetic) and the sympathetic divisions of the autonomic nervous system. The fibers of both divisions pass between the layers of the mesentery along with the blood vessels to the bowel wall. Here it ends in the myenteric plexus of Auerbach which is found between the circular and longitudinal muscle layers and the plexus of Meissner which is submucosal in position. Both contain ganglion cells pre and post ganglionic fibers of the vagus and post ganglionic fibers of the sympathetic system. In addition short fibers connecting one short segment of bowel with another are found. It has been postulated that the reflexes which control the propulsive intestinal movements operate through the intersegmental

fibers. Stimulation of the vagus (parasympathetic) fibers increases the tonus of the small bowel as well as its peristaltic activity. Stimulation of the splanchnics (sympathetic) fibers relaxes the intestine and diminishes peristalsis. In actual practice however the reactions observed in humans are not quite as simple as outlined.

There has been ample demonstration that the vagal effect is mediated by the production of acetylcholine. Injection of acetylcholine results in effects similar to vagal stimulation. Stimulation of the sympathetic fibers produces a substance similar to adrenalin in effect. These two substances are known as chemical mediators. The production of acetylcholine by cholineacetylase requires among other things the proper concentration of potassium and thiamin. An insufficient amount of either would interfere with the production of acetylcholine and as a result impaired peristaltic activity would occur. In addition the acetylcholine is inactivated rapidly by an enzyme found in nerve tissue which is called acetylcholine esterase. This latter is destroyed by a number of substances including Prostigmin. As a result the administration of Prostigmin has an effect similar to vagal stimulation because it prolongs and intensifies the effect of acetylcholine at the myoneural junction. Prostigmin would have little effect however if the acetylcholine is diminished in amount or is absent. It has been generally accepted that acetylcholine is needed for the nervous impulse of vagal stimulation to pass along the fiber and cross the synapse. Here it must be rapidly destroyed in order to permit the passage of the next impulse.

Adrenalin is produced by the sympathetic nerve with a resultant counteraction to the effects of vagal stimulation. The exact mechanism by which this occurs is not known. There is some evidence to suggest that it acts in some respects like an inhibitor of acetylcholine.

Watkins and Mann studied the motor response of spatially transposed intestinal loops. They exteriorized skin covered loops of jejunum and ileum and studied the effect of feeding upon the rhythmic contractions and their motor responses. It was found that these rhythmic contractions were constant for any given loop of intestine. The motor feeding response was found to occur more

rapidly in those loops which were nearer the pylorus than in the more distal loop. Changing the position of the intestinal loops with respect to their distance from the pylorus was found to produce rhythmic contractions in the transplanted loops in the same order as when the loop was in its original position. From this it was concluded that the rate of rhythmic contraction was a property of the loop itself and that it did not depend upon the relative position of the loop within the intestinal tract. The time of the feeding response was seen to depend upon the relative position of the loop with relation to the remainder of the gastrointestinal tract. The more oral the loop, the quicker did the motor response occur. Their observations supported the contention that feeding produces a wave of activity passing down the length of the small intestine.

The motor function is an extremely important function of the gastrointestinal tract. Intestinal movements are chiefly of two types—the rhythmic contractions and true peristaltic wave. The rhythmic contractions are thought to be myogenic in their origin. The smooth muscle of the bowel possesses inherent power of contractility similar to that of cardiac muscle. Peristaltic waves on the other hand are neurogenic in origin although they may occur after severance of their connections with the brain. Fainting the intestine with nicotine however completely abolishes peristaltic activity. From this it must be deduced that the intrinsic activity present in the bowel wall after section of vagus and sympathetics is due to the action of the myenteric plexuses. Peristalsis is subject to some regulation by the central nervous system under normal conditions. Emotional stimuli of various types acting from the higher centers exert an effect upon peristaltic activity. Functional bowel obstruction may be explained by a stimulation of the splanchnic nerves or sympathetic system regardless of cause.

Two to three thousand cubic centimeters of succus entericus are formed by the small bowel daily. Much of this in addition to the water and inorganic salts entering the alimentary tract is completely reabsorbed. Under normal conditions little water is lost to the body in the stool. This reabsorption takes place in the ileum and colon

and can occur only if the intestinal contents pass uninterruptedly down the gastrointestinal tract. It has long been known that the continued loss of secretions of the mucosa of the jejunum or ileum is compatible with normal health for long periods of time. Thirty Vella fistulas using shorter or longer areas of parts of the small bowel in which the succus entericus has been drained away have been made by numerous observers. No harmful effects have been observed. Herrin and Meek have demonstrated that when the small bowel is distended the secretion of succus entericus may become augmented that appreciable losses of electrolytes occur.

ILEOCECAL VALVE

This valve permits the small bowel contents to flow into the cecum but when competent prevents the reflux of cecal contents into the ileum. The exact mechanism by means of which this occurs is still not fully understood. When tightly closed the valve offers considerable resistance to the passage of material from the cecum into the ileum. The orifice opens rhythmically to permit a jet of fluid to escape into the cecum. Emotional excitement or the ingestion of food increases the frequency of these ejections. This is known as the gastroileal reflex.

The motor nerve to the ileocecal sphincter is sympathetic. Stimulation of this nerve results in a contraction of the sphincter and an inhibition of the wall of the ileum. Contrary to expectations the vagus nerve appears to have little or no effect upon the sphincter.

COLON

The colon differs from the small intestine in being much shorter and presenting a relatively fixed peritoneal attachment. Its walls are thinner than those of the small bowel and they are less muscular. The lumen of the colon particularly the right colon is much larger than that of the small intestine. The blood supply and vascular anastomotic channels are much less profuse than those of the small bowel. The propulsive peristalsis differs considerably. In the small intestine propulsive peristalsis is a progressive wave of gradual annular contraction and relaxation succeeding each other at frequent periodic intervals. In the

colon peristalsis is of the mass segmental type. It occurs at less frequent intervals moving the bowel contents suddenly and abruptly from one segment to the next. The function of the small intestine is primarily concerned with secretion and absorption of ingested material. Its secretory activity is far greater than that of the colon. The principal functions of the colon are absorption and storage. The secretory response of the small bowel to distention and circulatory stimuli is much greater than that of the colon. Because of this the character of the intestinal contents in each segment is very different.

The content of the small bowel is fluid. It contains more secretory substances and fewer bacteria. The colon content is semisolid or solid and it contains fewer secretory substances with the exception of mucus. The bacterial content is considerably greater in the colon and it increases markedly as it moves toward the rectum.

The physiologic rationale for a one stage operation on the right colon is based primarily upon the fluid content as well as the lower bacterial count of this portion of the gut as compared with the left colon.

Obstruction of the colon may produce a reflex increase in tonicity of the ileocecal valve. This may result in a closed loop type of obstruction. Clinical experience fails to support the contention that closed loop obstruction due to obstructive lesions of the left colon occurs with the frequency so often written about. Clinical evidence at Grace Hospital in Detroit rather supports the views of most radiologists that the ileocecal valve is completely competent in only a small percentage of cases—less than 20 per cent according to Grace Hospital statistics. It is only in this small percentage of patients that one need consider the possibility of cecal blow out due to left colonic obstructions.

In the colon the fibers of the outer muscular layer are gathered into three longitudinal bands. These are the tenia coli. Since they are shorter than the underlying layer they draw the bowel into pockets known as haustra.

The myenteric plexuses of Auerbach and Meissner are similar in position to those in the small bowel. The extrinsic nerve supply differs how-

ever from that of the small bowel. The vagal innervation terminates within the first half or third of the transverse colon. The remainder of the colon and rectum receives its motor nerve supply from the nervi erigentes. These pelvic nerves come from the second, third, and fourth sacral segments. The inhibitory fibers to the entire colon are derived from the sympathetics. These nerves are thought to exert a continuous inhibitory action. Section or blocking of them results in increased colonic activity. Absence of myenteric plexuses results in a functional type of paralysis producing a localized obstruction to the passage of intestinal contents. As a result the colon proximal to this dilates. The best example of this is Hirschsprung's disease.

TRANSIT TIME FROM MOUTH TO ANUS

The speed with which ingested material passes through the gastrointestinal tract is extremely variable. It depends upon the type of food ingested and its physical as well as chemical constituents. There are also individual variations in the activity of the intestinal musculature which may be greatly influenced by nervous and emotional factors. The absorptive function of the colon also varies and as a result the consistency of the colonic contents will differ. The degree of filling of the intestinal tract is also said to play a role. After a fast or with an empty gastrointestinal tract the food may move more rapidly only to be stored longer in the pelvic colon and rectum.

Best and Taylor report that fats and carbohydrates stimulate the movements of the small bowel the former by the formation of glycerin and soaps. The usual time required for the passage under normal conditions averages 24 hours. The food begins to leave the stomach shortly after being eaten. It then moves through the duodenum and down the jejunum fairly rapidly. Its progress down the ileum becomes slower, and it tends to accumulate before the ileocecal valve. From here it passes into the cecum in jets. It normally requires $2\frac{1}{2}$ hours for the food to reach the cecum. At the end of five hours the food has generally reached the hepatic flexure or midtransverse colon. In six hours it may be found at the splenic flexure.

Under normal circumstances in the average human the actual evacuation of the excreta occurs from 16 to 24 hour after ingestion of the food.

The rate of movement may be greatly increased or inhibited by the use of parasympathomimetic or sympathomimetic drugs respectively. In the former group are acetylcholine, pilocarpine,

Urecholine and Mecholyl. Adrenalin is in the latter group. In addition to these ganglion blocking drugs such as tetrathylammonium, Banthine and I to Banthine are inhibiting to all types of intestinal motility. Atropine has been widely used for many years to reduce the tone of the intestinal musculature and relieve spasm.

OBSTRUCTION OF THE ESOPHAGUS

WILLIAM A HUDSON, M.D.

Since the esophagus is an organ for swallowing and constitutes a passageway by which food is brought from the mouth into the stomach obstructions at this point make their presence known at a very early stage. A painstaking history of the character and onset of symptoms as well as their severity and distribution will facilitate an early diagnosis of their cause and when combined with the observation of such physical phenomena as nutrition, the drooling of saliva, cyanosis and distention of veins of the upper chest and neck will give a good idea of the general location and character of the lesions as well as an idea of the laboratory procedures best suited for their further study. I have never encountered an incident which justified throwing caution to the winds and rushing into some ill planned program for the treatment of esophageal lesions. Although there are certain occasions for urgency for example rupture of the esophagus or obstruction of the esophagus by a very large object with tracheal compression which may require immediate action to save a life even then a routine program will operate to the advantage of the patient.

The similarity of the symptoms in the various diseases of the esophagus can be very confusing. This similarity is a challenge to the diagnostician to sort out the symptoms with regard to their onset and their relationship both to each other and to the duration of the illness. Pain and difficulty in swallowing are cardinal symptoms of esophageal disease but variations in the onset, duration and extent of the disability caused by the numerous diseases which can affect the esophagus may be

confusing at first sight. It should be borne in mind that such complaints as soreness, gurgling sound and a sensation of something lodged in the throat and regurgitation of food will fall into the category of difficulty in swallowing and should lead one to a careful study of the condition of the esophagus.

In a considerable percentage of cases where there is a sensation of having something lodged in the throat the object is arrested momentarily in the pharynx or esophagus only to pass on without inflicting more than temporary discomfort. However, this knowledge should not lead one to disregard the fact that many objects do lodge in and perforate the wall of the esophagus thereby exposing the patient to the danger of peri esophageal or mediastinal infection and its most serious consequences.

SYMPTOMS

1. Pain in the neck or along the sternal area such as is found when a foreign body is present.
2. Substernal discomfort which may be described as a pain or as a pressure fullness.
3. Difficulty in swallowing food lodges momentarily or for longer periods.
4. Regurgitation this may occur soon after eating or at stated intervals.
5. Vomiting of blood.
6. Gurgling sounds on swallowing or on palpation.
7. Subcutaneous emphysema, perforation of the esophagus or a flapslike tear of the mucous membrane.

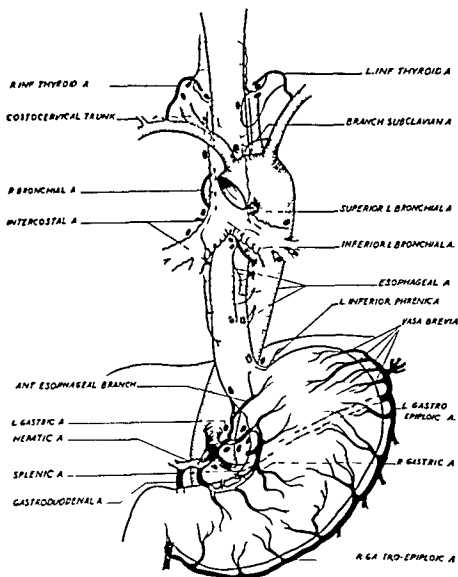


FIG. 29 Normal anatomy of the esophagus and stomach. Note the relationships to the aorta.

- 8 Loss of weight
- 9 Respiratory distress—compression of the trachea or bronchi
- 10 Hoarseness
- 11 A constant desire to swallow or expectorate saliva
- 12 Coughing after one or two draughts of liquid (high obstruction is indicated by this)
- 13 Foul breath
- 14 A mass in the neck reduced in size after regurgitation

DIAGNOSTIC PROCEDURES

Any patient who develops discomfort to such a degree that he comes for advice or relief should be given a general physical examination with particular attention paid to the condition of the nose, mouth, throat, pharynx, larynx, and pyriform sinuses. Blood counts and urinalysis are routine examinations which should never be omitted. In addition a preliminary fluoroscopic study of all parts from the head to the pelvis should be made. It is desirable as a rule to obtain postero-anterior and lateral X-ray films of the involved parts. As a rule metallic objects are readily demonstrated

in postero anterior and lateral films. Additional fluoroscopic and radiographic studies using contrast media may be desirable. The contrast media most commonly used are liquid barium, barium capsules and iodized oil. The particular medium and the time and method of its use should be determined in each individual case according to the indications at the time of the study. This combination of studies affords the best opportunity for one to observe variations in the contour and function of the parts under investigation. Frequently such variations are the best clue to the site and character of the disease or abnormal condition present. Radiographic studies with some sort of a film record are most desirable in all cases. The film serves as a permanent record for future reference and it is a valuable protection for the doctor from a medico-legal standpoint.

Esophagoscopy for confirmation of the findings demonstrated in the previous investigation may be the means of finally establishing a diagnosis. Direct inspection of the esophagus and microscopic examination of tissue specimens are of inestimable value. In the management of esophageal lesions, esophagoscopy is of diagnostic and therapeutic value.

The potential seriousness of all esophageal lesions and their complications makes an accurate diagnosis and considerable forethought in the choice of therapeutic agencies and measures imperative. Many esophageal lesions were diagnosed correctly and treated successfully before the introduction of X-ray and esophagoscopy; however, the proper use of the X-ray and the esophagoscope has led to more accurate diagnosis and more successful treatment of a greater number of esophageal lesions than was possible prior to their introduction. It is my firm conviction that, with the rarest exception, no man is justified in proceeding with any form of instrumentation of the esophagus before proper X-ray studies have been made.

OBSTRUCTIVE DISEASES AND THEIR TREATMENT

There are a number of disorders of the esophagus capable of producing obstruction. In addition, there are many disorders of the esophagus which, although not producing obstruction *per se*, never-

theless have the effect of obstructing the esophagus by making swallowing so painful that it becomes virtually impossible to use this organ as a means of conveying food to the stomach. For this reason, such conditions should be considered in any discussion of esophageal obstructions.

Esophagitis

Inflammation of the esophagus is not uncommon and is characterized by variable degrees of redness and swelling of the mucous membrane with loss of vascular markings. Edema may be so great that it produces folds in the mucous membrane and diminution in the lumen of the esophagus, thereby obstructing it. Acute inflammation may result from extension of pharyngeal or laryngeal infection into the esophagus. The submucosa may be involved and suppuration may result. Successful treatment of such lesions may be best accomplished through the control of the primary infecting process.

Tuberculous esophagitis is usually a complication or an extension of laryngeal tuberculosis and may be characterized by ulceration and bleeding. Swallowing of food may be so painful that a consequent loss of weight is the ultimate result. Tuberculous esophagitis can best be treated by using the same hygienic measures, antibiotics and chemotherapeutic agents that have been found to be effective in the treatment of laryngeal and other tuberculous infections—rest, therapy, acceptable food, streptomycin, PAS, isonicotinic acid hydrazide and their companion agents.

Inflammation of the esophagus from actinomycosis and other fungi occurs infrequently. The diagnosis of such lesions is made through the identification of the fungus involved. Such lesions are best treated through the use of the following drugs: iodides, which should be used regardless of other adjuncts; sulfonamides; penicillin; streptomycin; Stilbamidine. Many doctors contend that penicillin is the best drug to use in the management of this type of esophageal lesion. Thyroid in oral doses of 1 to 1 1/2 grams daily has been reported to be of some benefit. X-ray and radium therapy have been advocated and are said to be advantageous. There are many who feel that surgical drainage, when necessary, and the use of

iodides with the administration of penicillin are preferable methods. The administration of iodide is found to be of great value in many of the mycotic infections of the esophagus. Their use is not without danger, however, particularly when given to those patients who have become acclimated to them. Such patients not only may fail to improve on iodide therapy but they may become rapidly worse.

Two methods have been described for the administration of iodides. The slow method consists of giving 3 drops of saturated solution of potassium iodide three times a day, increasing 1 drop daily until the patient receives 20 drops three times a day. The dosage is then reduced gradually to the initial dose and again increased to 20 drops three times a day. This is the preferable method. The more rapid method consists of beginning with 5 drops three times a day and increasing 1 drop each dose or 3 drops a day. The dosage may be increased to 100 drops three times a day. In some cases sodium iodide is given intravenously in a daily dose of 1 gram.

Esophagitis complicating typhoid fever is much less frequent today than it was formerly. Typhoid fever is best controlled through preventive measures, good sanitation, typhoid vaccines and the use of chloromycetin.

Diphtheria of the esophagus is also rare since the process of immunization has reduced the incidence of diphtheria. Its diagnosis can be made through the identification of the diphtheria organism in smears and cultures from the lesions.

Esophagitis due to chemical irritation, iodine, hydrochloric acid and other corrosive agents is readily recognized both from the case history of ingestion of such an agent and from the usual excoriation of the buccal mucous membranes by the agent. Immediate treatment of such lesions should be carried out with the utmost gentleness, using a neutralizing agent at once.

One may use weak acetic acid or mild alkali in the case of alkali or acid burns, respectively, and starch water may be used for iodine burns. The use of penicillin and chemotherapeutic agents to control the infection will help to reduce the amount of scar which develops in the process of healing. I am certain that the ulceration will be

less extensive and that active dilatation of the esophagus can be initiated within the first week in many instances, thereby lessening the dangers of complete stenosis. The causal agent or disease must be treated in all cases of stricture of the esophagus. A narrowing of the esophagus which results from scar formed in the process of healing must be dealt with by gentle and careful dilatation.

One should be careful that he does not overlook evidence of any of the agents having entered the airway, larynx or trachea. If the larynx is obstructed, proper provision should be made to maintain ample airway. Tracheotomy may be a life-saving measure. The esophageal lesion should be treated with sterile olive oil and the early but careful passing of an esophagoscope of proper size for the aspiration of secretions and prevention of fusion of the esophageal walls. Dilatation of the esophagus should be done regularly, once or twice weekly, to prevent contracting scars from obstructing the esophagus. Fluids can be administered by vein if sufficient fluids cannot be taken by mouth. The use of penicillin and sulfonamide drugs lessens the danger of complete occlusion of the esophagus by contracting scars. Gastrostomy for feeding and retrograde bougienage are often necessary.

Chronic inflammation of the esophagus is usually associated with the results from the prolonged presence of foreign material, food or secretion in the esophagus. The symptoms are usually discomfort or a dull ache around the ribs, the sternal region or the back with varying degrees of difficulty in swallowing. Treatment consists of removal or correction of the cause of the retention of foreign material, food or secretion and then the use of penicillin and sulfa drugs. Any strictures resulting from such lesions should be treated by esophagoscopy and dilatation of the stricture.

Ulcers

Ulcers of the esophagus may be accompanied by pain on swallowing. If the ulcer is in the upper third of the esophagus, there may be regurgitation of blood and difficulty in swallowing. An esophageal ulcer may be caused by the presence of a foreign body of long residence.

Tuberculous ulcers are usually associated with

GASTRO INTESTINAL OBSTRUCTION

50

tuberculous laryngitis and pulmonary tuberculosis and should be treated with streptomycin at the same time the pulmonary or laryngeal lesions are being treated

Syphilitic ulcers of the esophagus are not frequent but such lesions should be proved by serologic and microscopic studies. Treatment is the same as that for systemic syphilis. In addition, those cases developing cicatricial obstruction should be subjected to dilatation or resection as indicated.

Peptic ulcers usually occur in the lower third of the esophagus and are accompanied by dull, boring pain that extends into the back. Radiologic studies may fail to demonstrate the ulcer. Esophagoscopy with biopsy gives the most convincing proof of the nature of the lesion. Treatment is the same as for gastric ulcer but resection of the ulcer-bearing portion of the esophagus with a portion of the stomach may be necessary.

Chronic nonspecific granulomas of the esophagus have been described and are characterized by the symptoms of obstruction. Prior to microscopic study they may be mistaken for carcinoma of the esophagus. Some of these lesions may be due to the ingestion of foreign bodies others may be secondary to local lesions within the mediastinum and in an occasional case no explanation can be found. Some cases respond to dilatation with a bougie others may become completely obstructed in spite of the measures applied and require resection of the involved portion of the esophagus or an esophagoplasty.

In addition to these types of esophageal ulcers ulceration may accompany malignancies and will be discussed with these neoplasms.

Strictures

A stricture is defined as a localized morbid narrowing of any passage of the body. The most common cause of the development of cicatricial stenosis of the esophagus is the ingestion of caustics or acids. The other factors which may cause narrowing or stricture of the esophagus are noted in the following outline and none should be disregarded. The cause of such stenosis is usually ascertained from the history. Two important features in the treatment of any stricture

are (1) ascertain and remove the cause, and (2) re-establish a passageway.

Any discussion of stricture of the esophagus if approached from its broadest aspect should include those localized narrowings of the esophagus which are intrinsic arising in the esophageal wall and those that are extrinsic or arising from without the wall of the esophagus.

Intrinsic stricture may be

- 1 Congenital or developmental in origin
- 2 Corrosive resulting from the swallowing of caustic acids or alkalis
- 3 Cicatricial resulting from the processes of healing of peptic ulcers, diphtheritic syphilitic, tuberculous, or nonspecific esophagitis
- 4 The result of healing of burns and scalds from ingestion of hot foods or other substances
- 5 The result of neurogenic disturbances
- 6 Caused by neoplastic tumors, including carcinoma and polyps

Extrinsic stenosis may be due to

- 1 Enlargement of mediastinal lymph nodes
- 2 Aneurysm
- 3 Spinal deformity with esophageal distortion
- 4 Compression or distortion by enlargement of the thyroid or tumors of the thyroid
- 5 Mediastinal tumors
- 6 Pericarditis or pleuritis adhesive or with effusion

Strictures most frequently multiple occur at the following sites: (1) the crossing of the left bronchus, (2) near the cricopharyngeal region and (3) the hiatus level.

The symptoms and local changes associated with stricture or esophageal obstruction are commonly described as being due to dysphagia. There may be regurgitation and a sense of fullness or distension after eating with a loss of weight. Complete obstruction may result from the lodging of particles of food at a narrow point. The stenotic area is usually pale and the scar may be very white, there may be retraction of mucosal surfaces or the area may be level with the adjacent surface or even raised as in the case of a keloid. The point of

narrowing although usually eccentric may be annular and at times there is pouching of the mucous membrane. Retention and stagnation of food in such areas may result in erosion and ulceration with bleeding.

Strictures due to burns with acids or other chemicals should be treated by dilatation under direct vision through the esophagoscope using graded bougies at intervals varying from once or twice a week to once a month or longer depending upon the severity of the obstruction and upon the response to treatment. The end result and the functional satisfaction will depend upon the extent and the density of the scar and the persistence and gentleness in application of the various procedures.

Esophagoscopy with sawing of webs with a string is dangerous because the possibility of the spread of infection throughout the mediastinum is too great. Areas of extensive scarring can be treated more safely by resection of the scarred portion of the esophagus and a re-establishment of the continuity of the passage by uniting the proximal and the distal ends of the cut esophagus or by uniting the proximal end of the esophagus with the stomach.

Perforation

One must never lose sight of the danger of perforation of the esophageal wall while passing any instrument. Such calamities have happened to the most experienced men but they occur most frequently with the less experienced and especially during blind bougienage. Most perforations occur either in attempts to pass an instrument through narrow points such as the cricopharyngeal region or cardia or during the removal of foreign bodies. Blind bougienage is never to be countenanced. In case of perforation immediate mediastinotomy with repair and continuous drainage, the antibiotic and chemotherapeutic measures are most urgent.

Perforation of the esophagus at instrumentation or because of other accidents is followed by *mediastinitis* and may result in death unless bold measures are instituted. Perforation of the subclavian artery with fatal hemorrhage into the pleural space has been reported. Esophagotomy is

less hazardous today than formerly because of the improved techniques and the use of chemotherapeutic and antibiotic agents. These agents enable us to combat infection and thus secure an early healing which was not formerly possible. Large objects and many pointed objects may now be removed more safely by esophagotomy than by other means. The majority of foreign bodies can be removed during esophagoscopy with an esophagoscope properly selected with regard to the size of the tube and with forceps suitable for grasping the kind of object to be removed. Extreme care must be exercised so that the object can be withdrawn without adding to the damage already caused by it.

Congenital Atresia

Atresia is defined by Webster as the absence or closure of a natural passage or channel of the body. This condition is an abnormality of the esophagus which is present at birth. The symptoms are dyspnea and strangulation from saliva entering the airway or from aspiration of feedings. These symptoms in any newborn child should lead to suspicion of such a defect. The careful insertion of a catheter with X-ray films as a record should give ample evidence of a blind pouch. Some advocate the use of iodized oil as a contrast medium. Immediate surgery is indicated; delay invites the further aspiration of secretion into the airway with tracheobronchial and pulmonary infection thus reducing the chances of a successful outcome. The best procedure is to establish a passageway from the mouth to the stomach by uniting the two segments of the esophagus through an end-to-end anastomosis. Any communication with the airway must be closed. The absence of sufficient esophageal tissue to permit the approximation of the two ends of the esophagus makes it necessary to resort to other means of establishing a passageway. A number of procedures have been used with varying degrees of success. Among the procedures that have enjoyed a measure of success are

- 1 The intrathoracic union of stomach or jejunum with the proximal esophageal stump
- 2 The formation of an extrathoracic esophagus through the use of skin tubes or by the

GASTRO INTESTINAL OBSTRUCTION

extrathoracic union of the stomach or jejunum with the proximal esophagus

The numerous cases which have been treated successfully attest to the value of these procedures for correction of this congenital defect

Cardiospasm

The term cardiospasm is applied to a pathological and clinical entity characterized by a narrowing of the esophageal passage at the diaphragmatic hiatus retention of food in the esophagus with chronic redundancy of the esophageal wall and chronic esophagitis Many descriptive names are used in classifying this condition Among these are achalasia idiopathic dilatation and phrenic spasm

The etiology of this condition is not fully known Jackson and Jackson described a pinchcock action of the crura of the diaphragm This dysfunction is associated with a disturbance in the action of the normal esophageal mechanism In many patients the presence of a neurotic factor is noted and in some cases, the passage of an esophagoscope or the use of a hydrostatic or aerostatic dilator may suffice to relieve the symptoms Usually the proximal dilatation or widening of the lumen of the esophagus does not disappear under such treatment Antispasmodics are used with a certain degree of success but one must not lose sight of the fact that periods of remission of the more severe symptoms may occur without any treatment The longer the disease has been present the more difficult it is to relieve by medical measures and esophageal dilatation may be relieved by surgical procedures Plastic operations of the type performed on the pylorus (Ramstedt type) have given relief in some cases It may be necessary to resect a segment of the esophagus and reanastomose the esophagus with the stomach On two occasions I have divided bands of scirrhous tissue without disturbing the muscular or mucosal layers and the patients have had complete relief There is another valuable plastic operation which enlarges the lumen by incising the full thickness of the esophageal wall into the lumen in the longitudinal plane Next the incision is resutured and the proximal end of the incision attached to the distal end thereby leaving a transverse widening of the

lumen at the point of closure I finally, one may resect a segment of the esophagus and join the proximal end of the esophagus to the stomach

It is my belief that the etiologic factors involved in the early stages of the disease are in all probability, much the same The lapse of time with retention of food the presence of infection increasing inflammation and the development of scar tissue with the drag of the biggy esophagus as it swings into the sulcus all aggravate the obstruction thereby causing the symptoms to increase in severity and become more persistent There may be a feeling of fullness or pressure within the mediastinum accompanied by actual soreness Eructation of gas and regurgitation of food commonly occur Large quantities of undigested food may be regurgitated when the patient stoops or lies flat As a result of the severity of such symptoms, surgical intervention may be necessary

Dysphagia

This term literally means difficulty in swallowing Two distinct types differing widely in etiology are described

Hysterical Dysphagia It is not uncommon for this diagnosis to be made but even in a patient who is known to be of an unusually nervous nature care should be taken to study all aspects of the complaint before accepting a diagnosis of hysteria Too many foreign bodies malnutrition and other diseased conditions have been overlooked because someone failed to study the problem Should no evidence of esophageal disease be demonstrated by the physical examination X ray studies and esophagoscopy followed by direct inspection of the esophagus then a diagnosis of hysterical dysphagia may be entertained The treatment should be the same as that for any other patient with hysteria On the other hand if a lesion is demonstrated treatment should be that for the specific lesion demonstrated

Dysphagia Lusoria This is a condition which is characterized by a difficulty in swallowing based upon an anatomic abnormality There may be a feeling of fullness or stiffness in the upper mid chest Radiologic studies reveal evidence of con-

pression or notching of the esophagus posteriorly at a level near the arch of the aorta. At esophagoscopy, narrowing and transmitted pulsations are observed in the posterior wall of the esophagus near or proximal to that produced anteriorly by the arch of the aorta.

The anatomic relationships which operate to produce this dysphagia are of embryonal origin. They are illustrated in the accompanying drawing (Figure 30) which shows an anomalous right subclavian artery arising from the left portion of the aortic arch and passing posterior to the esophagus. The esophagus is thereby caught between two large vessels. Treatment has been symptomatic except that more recently the anomalous vessel has been sectioned so that pressure against the esophagus may be relieved.

Other vascular anomalies such as double aortic arch which produces a vascular ring to encircle the

esophagus may also produce this type of esophageal obstruction.

Diverticulum

A diverticulum is a blind tube or sac branching off from a cavity or canal. Such blind sacs occur along the esophagus. Two recognizable types of diverticulum are described.

Pulsion Diverticulum The junction of the esophagus and the pharynx is a site of predilection for the pulsion type of diverticulum. The diverticulum occurring at this site are related to the anatomy of this part and are brought about by the relationship between the inferior constrictor muscle and the obliquely passing fibers of the cricopharyngeus as they descend upon the posterior wall of the esophagus to become longitudinal fibers. It is at this point when there is incoordination in the function of these related muscles that

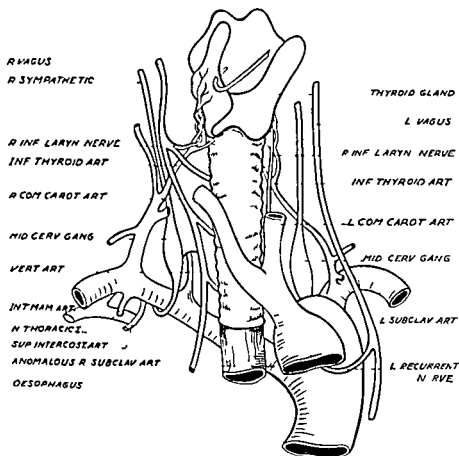


FIG 30 Anomalous right subclavian artery. Note the compression of the esophagus between the right common carotid artery of the anomalous vessel and the innominate artery on the right side.

pulsion diverticuli occur. They appear first as a small bulge which soon becomes a sac. As the sac enlarges its course is downward and it becomes distended with food. Its walls are composed largely of mucosa and submucosa. With increasing size the long axis of the sac comes to lie parallel to the long axis of the esophagus. On swallowing the sac moves up and down in the neck. As the sac grows it enlarges downward into the superior mediastinum where it may produce esophageal compression.

Instrumentation of the esophagus in the presence of a large sac becomes hazardous because of the danger of perforation of the wall of the sac. It is very likely that the instrument will be passed directly into the sac and through its distal wall instead of down the main esophageal channel.

Diverticuli of the esophagus may be complicated by the presence of peri-esophageal infection or by the presence of carcinoma in the sac. The presence of blood in the mucus expelled from the sac should lead to a suspicion of malignancy.

The demonstration of the presence of a distinct body neck and aperture into the esophagus through the use of contrast media at fluoroscopy and radiography makes a diagnosis of pulsion diverticulum almost certain. It is important that both postero-anterior and lateral X-rays be taken; otherwise a spherical dilatation of the esophagus may be mistaken for a diverticulum. In certain cases dilatations of this type occur proximal to the scar of previous operations or strictures.

The symptoms vary with the various stages of development. In the early stages when the sac is merely a bulge the only symptoms may be a feeling of fullness in the neck or the occasional lodging of a particle of food within the bulge necessitating a clearing of the throat. As the sac enlarges and more food is caught within it an unpredictable expulsion of food may occur at any time. The mixture of food and air within the sac may produce readily audible gurgling sounds upon swallowing or when pressure is placed upon the sac. Finally as the sac continues to enlarge obstructive symptoms occur. In the case of large sacs the symptoms are due to the downward drag of the heavy sac which causes the opening into the sac to become aligned with the proximal

esophageal lumen so that food passes directly through the opening into the sac. At the same time the esophageal passage is distorted in such a manner that food cannot pass down the esophagus into the stomach. During that period when the diverticulum has not yet produced major deformity of the esophageal passage and while food will still pass down the esophagus dilatation may relieve the obstructive symptoms but it does not delay materially the increase in the size of the sac. The danger of perforation of the sac by the passage of instruments must constantly be kept in mind.

The treatment of choice for pulsion diverticulum is the surgical removal of the sac. Although this should not be done too early when the sac is a mere bulge the ideal time for surgery in such cases is when the sac is still small enough so that no inflammatory circumferential adhesions have developed. At this time the aperture between the sac and the esophagus is not large; the neck of the sac is therefore relatively small and easily treated.

Two types of operation are commonly performed. Some surgeons prefer to dissect the sac free from its surrounding structures, cut it away across the neck and suture the defect—all in one operation. Others prefer to dissect the sac free from its surrounding structures and implant it into the wound in an inverted position when possible, or if the sac is too large it is brought out through the wound to lie upon the neck. The wound is then closed around it. The sac is resected at a later date and the esophageal opening is closed with sutures. The advantage of this latter procedure (two-stage operation) is that with a larger sac the danger of infection in the tissues of the neck is greatly reduced by sealing off fascial planes as a result of healing of the soft tissue around it. Eaks, which occur at the time the sac is cut away usually heal.

Other procedures such as inversion of the sac and cutting it off with a snare through the esophagoscope or twisting the neck of the sac and suturing it in place have been used in treating pulsion diverticuli but none are as free of complication or as satisfactory as either the one or two stage operation. The use of antibiotic aid in the

control of infection and renders the one stage operation the procedure of choice.

Traction Diverticulum A traction diverticulum is an outpouching from the esophagus due to adhesions and consequent traction. Traction diverticuli of the esophagus are usually asymptomatic and rarely require treatment. While they occur along the full length of the esophagus, they are more common in the middle and distal third of this structure. In the event that the sac grows to such a size that it causes symptoms, excision with careful repair of the esophageal defect is the procedure of choice. Antibiotics such as penicillin, streptomycin, and sulfonamide drugs should be used freely in any instance in which surgery of the esophagus is undertaken.

Herniations at Diaphragmatic Level

Herniations at the diaphragmatic level are of two main types. On the one hand the hernia may occur through the diaphragm *per se* by a separation or break in its musculature anywhere through out its expanse. Practically, however, because of the protective action of the liver, such herniations are most common on the left side. A second type of hernia at the diaphragmatic level occurs as a result of protrusion of peritoneum and intestinal contents through the normal anatomic openings in the diaphragm. The inferior vena cava, esophagus, and aorta pass through the diaphragm by means of separate apertures. In addition, an aperture is found just behind the xiphoid and the residual pleuroperitoneal canal through which the hernia of Bochdalek passes.

Hiatus Hernia This is the type of hernia that passes through the esophageal opening. It may be congenital, acquired, or postoperative. The commonest site for this type of hernia is between the vertebral body and the posterior wall of the esophagus because of the loose areolar tissue present at the esophageal opening. Repeated episodes of increased intra abdominal pressure may cause this loose tissue to give, and this results in the herniation of the stomach or small bowel into this space. As a result, a peritoneal sac is formed by the protrusion into this hiatal space. Such patients may complain of difficulty in swallowing, substernal distress, or symptoms attributable to cardiac dis-

ease. If strangulation of a loop of bowel occurs, symptoms of acute intestinal obstruction may supervene rapidly. A diagnosis in all such cases can be established by an upper gastro intestinal X ray using barium as a contrast medium. The management of lesions of this type is purely surgical. The repair of this type of hernia is preferably trans thoracic, usually through an incision at the level of the ninth rib or the intercostal space. At operation, the hernia is reduced, the hernial sac is obliterated, and the defect in the diaphragm is repaired. Although it is possible to repair these hernias transabdominally, if strangulation of the bowel is expected, or if the condition of the patient is such that bowel resection is anticipated, the thoracic approach is most desirable. In those cases in which bowel resection may be anticipated, or if the condition of the patient is poor and simple reduction of the incarcerated bowel is the only procedure practiced, a transabdominal approach may be used. The transabdominal approach, however, is more difficult and the chances of obtaining a satisfactory closure of the diaphragmatic defect are less good.

The postoperative type of hiatus hernia is the result of the popularity of esophagogastrostomies for esophageal carcinoma and the thoraco abdominal operations for upper abdominal lesions. Hernia of the diaphragm may develop if the stomach is not properly sutured to the diaphragm or the diaphragmatic opening is not properly closed after such surgery. In some cases, incorrect placing of the sutures may leave such a large defect in the diaphragm that herniation of the abdominal viscera into the chest results. The treatment of such hernias consists of a reduction of the hernial sac or reduction of the hernia and repair of the defect. With the increased popularity of vagotomy performed transthoracically for the treatment of duodenal ulcers, there should be an increase in the incidence of post surgical hiatus hernia. During the delivery of the lower end of the esophagus for dissection of the vagi, there is a tendency to loosen the attachments of the esophagus to the pillars of the diaphragm. If this defect is not repaired, it is possible for herniation to occur at this point.

Diaphragmatic Hernia Hernias of this type

pass directly through the diaphragmatic muscle and not through pre formed openings. The traumatic hernia of this type is generally the result of a sudden blow to the lower chest or upper abdomen or the result of a compressing accident. The clinical picture may be the same as that of a hiatus hernia. However unlike the hiatus hernia this type of hernia has no sac. They are not therefore true hernias but are classified as such by common usage.

The splenic flexure of the colon, the fundus of the stomach, spleen or small bowel may be found within this type of hernia in the thorax. In fact any or almost all of the abdominal viscera may be displaced into the thorax.

The diagnosis may be made certain by radiographic study after the ingestion of a barium meal with the patient in the Trendelenburg position. The treatment consists of the reduction of the hernia and closure of the defect in the diaphragm.

Neoplasms

The neoplasms of the esophagus which may cause obstruction fall into three large groups. These are (1) cysts (2) benign tumors and (3) carcinoma.

Cysts Cysts of the esophagus are usually congenital in origin. They may contain glands which produce secretions of a gastric nature which cause these cysts to become very large. The symptoms are the result of pressure on the adjacent organs as well as complications due to infection. Obstructive symptoms may occur because of compression of the esophagus. The method of treatment is the surgical removal of the cyst.

Benign Tumors Benign tumors of the esophagus occur infrequently and are curable. The symptoms in this case are similar to those encountered in any obstructive lesion of the esophagus. Dysphagia may not appear as early with benign lesions as with carcinoma. Radiologic studies with contrast media present the most characteristic indication of the benign nature of the lesion—the elasticity of the esophageal wall. Unless there is ulceration the outline of the tumor is as a rule smooth with a break in the continuity of the outline appearing only at the upper margin of the negative shadow.

These tumors lend themselves to surgical removal. Reports are found indicating the successful removal of pedunculated tumors through the esophagoscope with the use of a snare. Others have been removed successfully through a trans-thoracic surgical approach with the esophagus being opened and the tumor excised. The esophagus is then repaired according to the fundamental rules for repair of this structure.

We have identified the following types of tumors through pathologic diagnosis:

- Aberrant thyroid
- Adenoma
- Benign giant cell tumor
- Cyst
- Fibroma
- Hemangioma
- Ictenoma
- Lipoma
- Liposarcoma
- Mycoma
- Myxofibroma
- Neurofibroma
- Osteochondroma
- Papilloma
- Polyps
- Unclassified benign tumors

Carcinoma Carcinoma of the esophagus is a devastating disease which occurs most frequently in men and women past middle age. The most common sites at which it occurs are in order of frequency, middle third, lower third and upper third at or near those natural points of narrowing in the esophagus. The incidence in the male and female varies in different series of cases, but about 75 per cent of all carcinoma of the esophagus occurs in the male. There is little variation in the incidence in the white and colored races. The distribution of the lesions in the esophagus also varies but about 5 per cent of the lesions occur in the cervical esophagus, 13.5 per cent in the upper third, 42.4 per cent in the middle third and 38.5 per cent in the lower third. Multiple lesions of the esophagus have also been reported. The esophageal carcinomas are classified histologically as follows:

1	Squamous	66
2	Adenocarcinoma	17.2

3 Undifferentiated	4.0%
4 Unclassified	21.3%

The squamous cell carcinoma may appear in any portion of the esophagus while the adenocarcinoma appears in the distal portion of the esophagus.

The symptoms of the disease are the same as those encountered in any obstructive process. The first symptom is usually that of a conscious difficulty in or interference with the act of swallowing. The severity of the difficulty is dependent upon the degree of esophageal narrowing and may be greater or less from time to time depending upon the extent of local inflammation and swelling of the mucous membrane. The character of the food taken at any time will influence the symptoms. Coarse foods may block the esophagus at the point of narrowing and the patient may be unable to swallow until the food which blocks the passage has been removed or dissolved. Liquids will usually pass until obstruction is complete. Bleeding occurs as a later symptom and may be severe when ulceration has taken place or after trauma to an otherwise intact mucous membrane. With increasing obstruction regurgitation or vomiting will sooner or later occur. Foul odor occurs when food accumulates and undergoes putrefaction. Pain is a variable symptom; the pain may be dull aching and in the back or substernal but if the carcinoma should involve the intercostal nerves pain may be severe and sharp and may radiate to distant areas. Weakness is a frequent symptom. Hoarseness is not uncommon and may result from the aspiration of food or liquid into the larynx and trachea from the pharynx or from involvement of the recurrent laryngeal nerve by the carcinomatous process.

Cough and sputum accompanied by fever are late symptoms which may result from aspiration of food with accompanying pulmonary infection introduced through the foreign material in the airway or with the spilling of esophageal contents through perforations into the airway. Such perforations usually occur late in the disease but are not uncommon.

Swelling of the neck with edema and congestion of the blood vessels of the upper part of the chest, shoulders, neck and head may be seen as a

result of the obstruction of the superior vena cava by the carcinoma or through pressure produced by the involved regional lymph nodes.

A diagnosis of carcinoma of the esophagus is dependent upon a careful study of the esophagus by means of X ray with the use of contrast medium being the one in most common use. This should be followed by esophagoscopy studies noting any deviation from normal in the appearance of the longitudinal folds of the mucous membrane. It is important to note any zones of fixation of the wall or any deformity in the contour of the lumen of the esophagus. Tissue for microscopic studies should be obtained from any masses or areas of induration or ulceration.

It is particularly important to realize that adenocarcinoma involving the lower esophagus not infrequently is the result of direct extension from a primary adenocarcinoma from the stomach. Primary adenocarcinomas involving the posterior wall of the stomach are extremely difficult to diagnose radiologically. The first inkling that such a lesion is present may be the development of dysphagia or difficulty in swallowing or an obstruction of the lower esophagus. Esophagoscopy examination readily discloses the carcinomatous nature of the lesion and biopsy confirms the fact that it is adenocarcinoma. In many of these patients the true diagnosis of primary carcinoma of the stomach with direct extension to the esophagus is not made until the time of surgery. Unfortunately in many cases when this occurs the primary malignancy of the stomach has already extended far beyond the confines of curative surgery. The best that one can hope for under such circumstances is palliation. The treatment of carcinoma of the esophagus was formerly symptomatic with bougienage and esophagoscopic dilatation in order to maintain a passageway as long as possible. X ray therapy has been used extensively but its curative powers were and are limited. As swallowing becomes more and more difficult a gastrostomy was generally established for feeding purposes. Torek and many others have established beyond a doubt the curability of carcinoma of the esophagus by surgical means. The attendant surgical problems have been so adequately solved that morbidity and mortality rates have been reduced considerably.

I frequently even in cases in which metastasis has occurred resection of the carcinoma or the by passing of the carcinoma with re establishment of a continuous passageway is preferable to gastrostomy or esophagoscopy with blind bougienage for maintenance of a passageway. One need only study the many reports in the literature to see the propriety of surgical removal of carcinoma of the esophagus.

Foreign Bodies

Many types of foreign bodies producing obstruction of the esophagus have been reported. Foreign bodies present a far different problem when they are in the esophagus than they do when they are distal to the esophagus. The tendency of foreign bodies having once entered the stomach to pass without difficulty through the gastrointestinal tract and be excreted through the rectum is well known. In the esophagus however arrest of such a foreign body may lead to very serious consequences. It is surprising to note that the size or shape of the foreign body has little or no relationship to whether or not it becomes arrested in the esophagus. Innumerable cases of swallowed foreign bodies excreted through the rectum or removed from the stomach or gastro intestinal tract have been reported since the Middle Ages. One of the earliest records was that reported by Gross who mentions that in 1502 Florin Mathias of Bradenberg removed a knife 9 inches long from the stomach of a 36 year old man who recovered successfully. When one considers the varied types and considerable size of those foreign bodies swallowed by psychotics which have passed through the esophagus with ease it at times becomes difficult to understand why foreign bodies of much smaller size become arrested.

A considerable proportion of foreign bodies which reach the stomach pass through the remainder of the gastrointestinal tract and as a result a conservative attitude is generally adopted with regard to their management. In the esophagus however sound medical principles dictate that any foreign body which has become arrested must be removed as soon as its presence is recognized. A major reason for this is that the chances of eventual spontaneous passage of such arrested

foreign bodies are usually not good. Furthermore the edema secondary to the local trauma not only tends to retain the foreign body but also makes eventual manipulation much more difficult. And in addition if perforation of the esophagus occurs as a result of the foreign body the surgical situation becomes extremely serious and the prognosis much worse.

There are many types of foreign bodies which are likely to be encountered in the esophagus, Jackson and Jackson list the most common in order of frequency as follows:

- 1 Bones
- 2 Food
- 3 Coins and discs
- 4 Safety pins
- 5 Straight pins
- 6 Buttons and wood
- 7 Toys and whistles
- 8 Dental plates
- 9 Nuts seeds shells
- 10 Animal shells
- 11 Hardware
- 12 Jewelry
- 13 Minerals
- 14 Ammunition
- 15 Hairpins

Aside from the ingestion of foreign bodies which are radiopaque and may be visualized by X ray obstruction of the esophagus may occur as a result of impaction of food. In such cases a survey film of the esophagus will not demonstrate the obstructing mechanism. In many of these cases obstruction of the esophagus occurs as a result of the impaction of the food in an already partially obstructed esophagus. This partial obstruction may be due to a benign tumor an early carcinoma or a stricture of the esophagus of many years duration. Of the various foods which occasionally obstruct the esophagus meats are by far the most common offenders. Less frequently fibrous vegetable products like persimmon skins, celery and coconut may be found as a causative factor. The patients in whom food produces an obstruction of the esophagus are generally elderly individuals or those with dentures who do not chew their food adequately.

Among the uncommon types of obstruction of the esophagus was a case reported by Mehmed and March of an obstruction of the lower esophagus resulting from the ingestion of Serutan. This is a hygroscopic substance which is a hydrogel obtained from *Platycodon ovata* and extracted from sugar beets from which the roughage has been removed. The mass producing the obstruction of the lower esophagus measured 17 by 29 mm and was of a rubbery consistency partially coated with the barium which was given in order to detect the type of obstruction and its level. Complete relief was obtained in this case after emesis of the foreign body.

The majority of foreign bodies which do not pass quickly into the stomach become caught in the cervical segment of the esophagus. This is caused by the fact that the strong propulsive pharyngeal muscles are able to force the foreign body thus far but the less active esophageal musculature is unable to propel it downward into the stomach. In addition the mucosa in this area is more mobile over the deeper layers than elsewhere in the esophagus.

Foreign bodies which transverse the cervical portion of the esophagus generally pass easily on into the stomach but may lodge at the level of the aortic arch at the level of the heart or at the cardia these being natural points of narrowing. As a result arrest of the foreign body in the thoracic or abdominal portion of the esophagus in an adult should always suggest the possibility of a tumor congenital anomaly or a stricture at the point of arrest. Strictures characteristically may be present for many years and produce no symptoms only to cause complete obstruction when a large foreign body such as a meat bolus lodges against them.

Some types of foreign bodies may remain in the esophagus for a long period of time without producing changes. Sooner or later however erosion of the esophageal wall occurs as a result of pressure. There may then be peri esophageal inflammatory changes which may extend to the pleura. There are many cases in which suppuration occurs with the formation of a fistula which discharges externally. Stricture very often develops at the site of lodgement of a foreign body if the

foreign body is not removed within a reasonable period of time.

The presence of a foreign body in the esophagus is usually diagnosed with the aid of a carefully taken history. The subjective changes complained of by the patient and the sensation of a difficulty in swallowing are of great importance. The radiologic examination may be conclusive in those cases in which the foreign body swallowed is radiopaque. In most cases there are some subjective sensations of a foreign body and often pain. In an occasional case there may be little or no sensation and the patient may not give a history of having swallowed a foreign body because the incident in which this occurred escaped his attention. The X ray will clearly show the presence and location of radiopaque foreign bodies but in the case of a radiolucent object the survey film of the esophagus will be negative. The swallowing of liquid barium or a capsule filled with dry barium will aid in demonstrating the level of the obstruction. The treatment for foreign bodies in the esophagus is always removal at the earliest possible moment. Whenever possible this should be accomplished through an esophagoscope. When this cannot be done esophagotomy with removal of the foreign body and closure of the opening in the esophagus is the procedure to be followed.

Traumatic or Inflammatory Obstructions of the Lower Esophagus

Inflammation of the lower esophagus followed by stenosis may be produced by the reflux of gastric juice associated with vomiting and gastric or intestinal intubation. This inflammatory stenosis may be explained by the development of esophagitis which is followed by ulceration and stenosis. This is the result of the action of the acid peptic gastric juice on the lower esophageal segment. It has been demonstrated that the irritation of the esophageal mucosa by gastric contents may produce esophagitis. Clinical evidence of reflux of gastric contents into the esophagus has been established. Reflux of barium from the stomach into the lower third of the esophagus does occur but it does not fill the entire esophagus. This reflux is most commonly found with gastro-intestinal disease. Vomiting or the passage of a Levin tube or

both were the preliminary cause in approximately 50 per cent of the cases. The increased intragastric pressure as a result of a pressure dressing on the upper abdomen or gastric distention, may cause a reflux of gastric juice through the esophagogastric junction which was held partially open by the Levin tube or by any long intestinal decompression tube.

Recurrent vomiting and gastro intestinal lesions may have been present for many years without esophagitis. Postoperative gastric retention and intestinal intubation or gastric intubation have been considered as being the precipitating agents which initiate the process. Stenotic esophagitis occurs in both sexes and in any age group. The diagnosis can only be made by radiologic examination with barium or esophagoscopy study.

Difficulty in swallowing particularly following intestinal intubation or gastric intubation should make one suspicious of an esophageal stenosis. The majority of benign strictures and stenotic areas may be found in the lower half of the esophagus chiefly in the terminal third. This location coincides with the acid peptic concept of its origin because this area is the most frequently exposed to concentrated gastric juice.

Intubation in Management of Obstructions of Lower Esophagus

Tubes have been used in two different ways in the management of obstructions of the lower esophagus. In those cases in which the obstruction is incomplete a Levin tube or a mercury tipped

fine plastic tube may be successfully passed into the stomach and used for feeding purposes. The mercury tipped plastic tube can usually be passed through very small esophageal openings. This tube can be used to improve the nutrition of the patient prior to surgery. When used in nonsurgical patients these tubes may be permitted to remain *in situ* for long periods of time. This method is simple and infinitely preferable to gastrostomy as a feeding mechanism.

The mercury weighted tube may be used to dilate the lower esophagus obstructed by cardiospasm or achalasia. According to a number of reports it appears that about 70 per cent of the cases are completely relieved by one course of treatment with the hydrostatic bag. Of these the recurrence rate is said to be approximately 30 per cent. In those that recur most of the patients may be successfully relieved by subsequent dilatation. In approximately 5 per cent of the cases in this latter group hydrostatic dilatation does not prove to be effective and surgical intervention becomes indicated.

Decompression of the esophagus is not obtained by means of a tube. Since it is the most proximal portion of the gastro intestinal tract the esophagus is readily emptied by vomiting. Reflux of gastric contents into the lower esophagus held open at the esophagogastric junction by a Levin tube or a long intestinal decompression tube may result in esophagitis. As a result ulcerative changes followed by stenosis may occur long after the removal of such tubes.

OBSTRUCTION OF THE STOMACH AND SPHINCTERS

Obstruction in the stomach may be due to atony of the stomach or to dynamic spasm of the stomach sphincters or it may be produced by the same factors causing small bowel obstruction. In cases of this type no purely mechanical obstructing mechanism is involved.

However, the most common cause of obstruction in the stomach is the mechanical lesion. In this group the lesions range from pyloric stenosis to volvulus of the entire stomach. Almost every conceivable type of mechanical obstruction may involve the stomach. Moreover the mechanical obstruction may be intermittent or continuous. This is particularly apt to occur at the pylorus where a ball valve action may be produced by a pedunculated prepyloric tumor. Although all available evidence suggests that cardiospasm should really be considered in the chapter on esophageal obstruction, we include it here because the questionable cardiac sphincter is in reality part of the stomach.

CARDIOSPASM

This lesion also called achalasia of the esophagus is characterized by a variable degree of dilatation of the esophagus proximal to the lower 1 to 4 cm. The degree of esophageal dilatation proximal to the point of obstruction varies with the duration of the process. In cases of short duration the esophagus dilates only slightly, whereas in long standing cases there may be tremendous esophageal dilatation. The muscular walls of the esophagus become markedly hypertrophied in an effort to overcome the obstructing mechanism. This reaction is believed to be functional in origin

since no evidence of stricture or hypertrophy has been demonstrated in the undilated portion. The microscopic finding of aganglionic areas in this undilated portion of the distal esophagus is responsible for the concept that the lesion is analogous to the obstructing mechanism of Hirschsprung's disease.

A failure of the diaphragmatic pinch-cock to open during deglutition has been suggested as a possible causative mechanism. Hurst has suggested that cardiospasm is the result of failure of the cardia to relax with the esophageal peristaltic wave. There is some experimental evidence suggesting that the sphincter is capable of functioning independently of the surrounding structures. Vagal stimulation has been shown experimentally to relax the sphincter, whereas sectioning of the vagus may result in temporary spasm. This is recognized clinically in the temporary cardiospasm following vagotomy as a treatment for duodenal ulcer.

In the vast majority of cases the symptoms produced consist of difficulty in swallowing, substernal discomfort or regurgitation. Such cases respond to neuropsychiatric treatment, sedation and the use of antispasmodic drugs. A failure to respond to these measures may require the use of dilating bougies. We as surgeons are not particularly concerned with these milder types of obstruction. In a small percentage of cases, certainly less than 20 per cent, the obstruction produced by cardiospasm may be of such a degree as to completely and persistently obstruct the cardiac opening of the stomach. Patients of this type literally

starve to death unless some surgical procedure is undertaken to correct the functional obstruction present. Various surgical procedures have been proposed to correct the obstructing mechanism.

Occasionally it may be difficult or impossible to determine prior to surgery whether the patient is suffering from achalasia or carcinoma of the distal esophagus. In cases of this type, with the patient in poor condition due to starvation a gastrostomy of the Jewway type may be desirable. This is a temporary measure permitting nutritional improvement so that more definitive surgery may be performed. In addition at the time of gastrostomy a digital and biopsy exploration of the cardiac sphincteric and lower esophageal areas may be carried out to reach an exact diagnosis.

STENOSIS AT THE CARDIA

This is the result of cicatricial healing of a peptic ulcer of the esophagus. It may be produced as a result of the action of gastric juice particularly in a hiatus hernia. This type of esophagitis is not infrequently associated with stenosing duodenal ulcerations. In its early stages the obstructing mechanism may be temporary and the result of spasm induced by irritation produced by the ulcerative process. In late stages cicatricial contraction results in a stenosing markedly narrowed and obstructed cardio esophageal junction. At this stage surgical correction is the only treatment possible.

Stenosis of the distal esophagus and cardia may result from the healing of caustic burns such as those induced by the ingestion of lye. Cases of this type were very commonly reported in the foreign literature on the subject. The treatment depends upon the degree of obstruction present. Where the process is incomplete dilating bougies have been successfully used. The surgical treatment is the same as that used for obstructions of the distal esophagus.

INFECTIONS

Systemic infections such as polymyelitis may produce a gastric ileus as part of a paralytic ileus involving the gastro intestinal tract. In cases of this type the stomach may dilate tremendously. This fluid filled tremendously dilated stomach constitutes a high intestinal obstruction on a

neurogenic basis. The loss of water and chlorides is tremendous in such cases. Dehydration and alkalosis rapidly ensue unless adequate replacement therapy is instituted. The passage of a Levin tube into the stomach with continuous suction thus keeping this viscus empty invariably results in recovery. The paralytic ileus in such cases is of relatively brief duration. The entire process may be adequately corrected within a week. The prevention of dehydration and the avoidance of electrolyte imbalance are of the greatest importance in such cases.

Tuberculosis and syphilis, particularly the latter, may produce a fibrosis in the stomach wall during its stage of healing. A localization of this process at the pylorus may result in a stenosis and obstruction.

FOREIGN BODIES

A high degree of mediogastric stricture may occur as a result of ingestion of caustic acids or alkalis. Numerous cases of this type have been re-



FIG. 31 Note tremendous gastric dilatation as a result of polymyelitis.



Fig 37 Mrs M P age 72 4 plus Kahn syphilitic pyloric obstruction

ported in the foreign literature in which stenosis of the media of the stomach or pylorus has been reported following the ingestion of 40 per cent hydrochloric or other corrosive acids. A high degree of obstruction at the pylorus or in the antrum occurs in all such cases that survive. Gastroenterostomy adequately corrects this problem.

Foreign bodies of all types have found their way into the stomach. They may be grouped into three types based upon their method of entrance or formation as follows: swallowed foreign bodies, bezoars, and those entering the stomach through a gastric fistula.

The vast majority of foreign bodies remain within the confines of the stomach or pass through it without producing any signs or symptoms. Obstruction at the cardia or pylorus may occasionally occur as a result of the ingestion of a foreign body; this is particularly apt to occur as a result of swallowing partial dentures which often become arrested at the lower esophagus producing obstruction at this point. The variety of swallowed foreign bodies is enormous. Nearly every con-

ceivable article from the most minute to the most incredibly large has been reported. Among the most unusual is the case report of Cloismadene who recorded the case of a young female hysteric who swallowed a cross 7 cm long, and with it a rosary 220 cm long with seven holy medals attached to it. Foreign bodies may remain within the stomach for many years without symptoms.

Bezoars are foreign bodies which increase in size by virtue of their ability to take on substance after being ingested. Among these are hair, shell, vegetable fibers, and medicaments like salol. Of these hair balls (trichobezoars) are by far the most common, with innumerable cases reported. One of the largest recorded was reported in *Lancet* in 1895. In this case a young girl presented at surgery a hair ball weighing five pounds and three ounces. (The earliest report appearing in the literature was that of Baudamant in 1779.) Vegetable and fruit fibers not infrequently form the nidus for the development of a phytobezoar. A stone weighing 75 grams was reported removed from the stomach of a painter who habitually drank small amounts of the shellac used in his trade. The shellac precipitated out forming a nidus for the further development of a large stone. One of the most unusual types of bezoars which passed down the gastrointestinal tract before becoming arrested weighed 470 grams; the nidus for this concretion was found to be a swallowed tooth.

Foreign bodies entering the stomach through fistula formation may occur following surgery or as the result of cholecystogastric fistula. In the former instance a foreign body such as a hemostat or a laparotomy sponge inadvertently left in the abdominal cavity may ulcerate into the stomach producing obstructive symptoms. Gallstones may produce obstructive symptoms after erosion into the stomach.

The management of any gastric obstruction resulting from the presence of a foreign body calls for gastrotomy with removal of the obstructing material. The procedures are relatively simple once diagnosis has been made.

CONGENITAL ANOMALIES

Double stomach or failure in the development of the gastric cavity are discussed in the chapter

on embryology (Chapter 2) Such anomalies rarely produce obstruction in adult life

NEUROGENIC CAUSES

Acute gastric dilatation is a postoperative complication which is generally easy to control. However occasional cases of gastric dilatation occur which do not respond to the usual form of treatment by gastric lavage or gastric suction. In such cases the constant and incessant vomiting may cause the death of the patient through dehydration. This complication following surgery does not seem to be caused by the anesthetic agent but it may follow any type of operative intervention upon any portion of the body. It most commonly occurs in surgery of the upper abdomen particularly biliary surgery. Joseph reported a group of cases in which the one common factor was enforced confinement in bed after surgery. In cases of this type early ambulation is a specific prophylactic. This disorder is considered to be an adynamic gastroduodenal ileus resulting from an inhibitory visceromotor reflex. This may be induced by trauma or painful stimulus during operation or by infection in the region of the pancreas. The afferent pathway of the reflex which inhibits gastric and duodenal motility is said to run with the sympathetic fibers. Blocking of the celiac ganglion by novocaine interrupts this reflex. Sympathetic commutic anesthetic agents such as cyclopropane are thought to sensitize the body for this reflex syndrome. Acute gastric dilatation is a reversible condition. Preliminary passage of the Levin tube will keep the stomach and duodenum empty and prevent the development of this complication. The postoperative passage of the Levin tube enables the stomach to regain its tonus by keeping it empty. As a result the motility which was lost by the activation of the reflex will be restored. Replacement of water and electrolytes counterbalances the secondary damage produced by the dehydration and disturbed acid base equilibrium resulting from the acute gastric dilatation. The associated paralytic ileus does not require separate attention but is a manifestation of the generalized process. It usually subsides with the disappearance of the acute gastroduodenal ileus.

An examination in such cases discloses a markedly dilated stomach. In some cases the dilatation is so extreme that it may fill the entire abdominal cavity. The gastric contents are usually found to consist of gas and a thin greenish or brownish black fluid which is odorless.

In the postoperative type the patients may be asymptomatic for the first 24 to 48 hours. Vomiting attributable to the anesthetic may be more marked than usual. The patient may complain of epigastric fullness. The upper abdomen is generally distended. There is usually no interference with the motor function of the small bowel. Pain is quite variable; it may be nothing more than a feeling of fullness or it may be extremely severe. The patient's breathing is rapid and shallow. The pulse is often rapid and feeble and the skin cold and clammy. In some cases collapse occurs early. This is particularly true when the acute dilatation is sudden in onset. These patients may become cyanotic show marked dyspnea and complain of considerable epigastric distress.

Enormous quantities of fluid may be removed from the stomach in these cases. This fluid usually contains little or no free hydrochloric acid but almost always contains bile. It is generally agreed that the fluid is made up of a mixture of digestive secretions. The amount present is far in excess of the normal volume secreted. This has led to the belief that gastric dilatation causes a great outpouring of liquid into the gastric lumen.

Paralysis agitans or postencephalic parkinsonism may be associated at times with severe intestinal distention. Attempts at intestinal intubation in such cases may be futile. Not infrequently the pyloric sphincter may be found to be in a condition of spasm so that the passage of an intestinal tube through it is doomed to failure. Our experience with gastric dilatation in case of this type has shown that the liberal use of antispasmodic drugs such as Tolserol will often convert a failure into ultimate success. Multiple sclerosis occasionally will be accompanied by gastro intestinal distention on a similar neurogenic basis. The treatment in all such cases is intubation and the liberal use of the newer ganglionic blocking agents such as hexamethonium or Pro Banthine.

NEOPLASTIC CAUSES

The most common cause of mechanical obstruction of the stomach is the presence of either a benign or malignant tumor within it. The most common benign tumor causing obstruction at the pylorus is the benign pedunculated tumor of the stomach which acts as an obturator to the pylorus. This may obstruct in one of two ways—either by functioning as a ball valve producing intermittent obstruction or by irritating such vigorous peristalsis that intussusception of the stomach into the duodenum is produced. In some cases a combination of the two processes occurs. The first case report of prolapse of a pedunculated gastric tumor through the pylorus with obstruction was reported by Breschet in 1816. He described a pyloric obstruction due to a polyp which arose from the lesser curvature 3 inches distal to the cardia and passed into the duodenum. Although many case reports of this type appeared in the European literature of the 1800's it was not until 1917 that attention in America was attracted to this subject by the writings of Gibb. Following this many other cases of benign tumors of the stomach causing intermittent obstruction were published.

Rigler reported that 24 per cent of a large group of gastric tumors noted pathologically were benign. This was supported by the observations of Dudley and his associates who found 22 per cent of all gastric tumors at autopsy to be benign. Of these benign tumors leiomyoma is the most common constituting from 25 to 40 per cent of all benign tumors reported. Although incidental findings at autopsy reveal most of these tumors to be asymptomatic gastric hemorrhage and gastric obstruction occur in an appreciable number of cases. This is usually the result of the common incidence of such tumors at the pyloric region of the stomach. Strangely enough obstruction was found to account for only 8 per cent of a group of 50 cases studied by Abrams. Bleeding was the most common manifestation being noted in 66 per cent of all cases. These tumors vary in size—from 1 to 2 cm. in diameter up to the 2 pound tumor reported by Blackland. A small tumor at the pylorus may be of sufficient size to produce obstruction to this viscus whereas a large one elsewhere may be

asymptomatic. Occasionally a very large tumor of this type may cause volvulus of the stomach by virtue of its weight. However small fibromas or myofibromas at the pylorus cause early gastric obstruction. These lesions are difficult to recognize clinically and may be missed radiologically. Benign tumors as a group should be suspected in any case in which the gastro intestinal symptoms are those of obstruction and the radiologic examination is not diagnostic.

Apart from leiomyomas of the stomach the other types of benign tumor are neurofibroma, fibroma, lipoma, osteoma, osteochondroma and myoma. The majority of these form rounded or flat tumors projecting into the stomach. Some on the other hand are pedunculated and cause obstruction by their obturator mechanism.

The treatment for all benign tumors of the stomach is subtotal gastrectomy. In those cases in which the tumor is pedunculated and projects into the peritoneal cavity simple resection of the tumor at its attachment to the stomach is sufficient.

It has been estimated that approximately 1 per cent of all malignant tumors of the stomach are sarcomatous. The following types are recognizable: (1) spindle cell sarcoma, (2) lymphogenous sarcoma, and (3) mixed round cell sarcoma. In their behavior this type of new growth may grow outward inward into the gastric lumen or infiltrate within its walls. The intragastric type may produce pedunculated tumors within the gastric lumen which may cause ball valve obstruction by passing through the pylorus. Strangulation of the prolapsed tumor may occur. Radical subtotal gastrectomy followed by irradiation for the lymphogenous type of sarcoma is the procedure most likely to succeed. In general the results obtainable with this type of tumor are better than with carcinomatous tumors. This is particularly true of the lymphogenous type of sarcoma where a 20 to 30 per cent 5 year survival may be expected.

The most common variety of malignant tumor obstructing the stomach is carcinoma and it is in the stomach that alimentary tract malignancy appears most frequently. The ulcerative type of carcinoma is the most common type seen. To prove the actual development of carcinoma in a



FIG. 33 Stenosing carcinoma of the stomach

crises is not infrequently obstructed by the overgrowth of fibrous connective tissue. A localized carcinoma of this type may be almost indistinguishable from hypertrophic tuberculosis of the pylorus, syphilis of the pylorus or cicatrizing pyloric stenosis. The antrum and pylorus in such cases show marked thickening caused by a dense white hard fibrous connective tissue which involves the submucosa and subserosa. The stomach wall has been reported to be an inch thick in such cases. Pedunculated polypoid carcinoma of the stomach may occasionally prolapse through the outlet of the stomach obstructing it.

The treatment for all types of carcinoma of the stomach producing obstruction is radical subtotal gastrectomy. Although the recurrence rate is high the obstructing process is relieved.

GASTRIC PILES

Intermittent gastric obstruction may occur as a result of the prolapse of redundant hypertrophic folds of gastric mucosa through the pylorus. Many papers have been written on this subject. Numerous descriptive titles have been applied to the phenomenon: prolapse of gastric mucosa, gastric piles, hypertrophic gastritis with prolapse and many others. Although these hypertrophic folds are generally asymptomatic they may on occasion produce intermittent obstruction due to prolapse. At times this obstruction becomes complete due to pyloric spasm around the prolapsed fold. This results in marked edema or even necrosis of the prolapsed fold.

ULCERS

The most common cause for pyloric stenosis is the cicatricial changes resulting from a healed pyloric or duodenal ulcer. This is the result of the fibroblastic proliferative changes resulting from the healing process in a duodenal or pyloric ulcer. The duodenum becomes fibrotic and contracted with each acute flare up of ulcer formation. The end result of many such exacerbations is the stenotic pylorus. The pylorus in such cases presents a hard cartilaginous mass. The overlying serosa is puckered or speckled and the duodenum distally is often apparently dilated. Not infrequently an acute exacerbation of a pyloric ulcer may produce a degree of edema causing complete

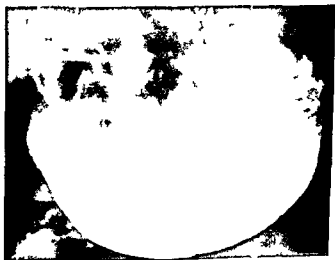


FIG. 34 Obstruction of pylorus due to carcinoma

previously benign ulcer is impossible. There is sufficient evidence, however, that 10 per cent of all ulcers considered benign by all known diagnostic methods prove ultimately to be malignant. This applies particularly to ulcerative lesions in the pyloric portion of the stomach. This type of carcinoma causes obstruction least often. The most common malignant type obstructing the stomach is the polypoid or fungating carcinoma. The large cauliflower-like masses readily obstruct the gastric outlet. Many of these begin as gastric polyps.

A third variety of carcinoma of the stomach is the type associated with marked fibrosis causing a leather bottle stomach. The pylorus in such



FIG 35 Note tremendous gastric retention as a result of stenosing pyloric ulcer

pyloric stenosis. Continuous suction and warm saline gastric washes via an indwelling Levin tube may often cause a rapid subsidence of the edema. As a result the obstruction is released. With proper medical management it may remain asymptomatic for a prolonged period of time. Many of these ultimately become completely stenosed by fibrous tissue. This presents an irreversible lesion. Gastric resection is the procedure of choice in such cases except for the poor risk or elderly patient with low acidity. In these cases gastroenterostomy for the elderly and gastroenterostomy with vagotomy for the younger poor risk patients are to be recommended.

POSTOPERATIVE

Many types of obstruction to the stomach occur after gastric surgery. These are reported in Chapter 24.

VOLVULUS

Volvulus may cause obstruction at the esophageal as well as the duodenal orifices. A rapidly pro-



FIG 36 Pyloric obstruction as a result of ulcer

gressive and fatal condition results unless early surgical corrective measures are instituted. A common type of volvulus occurs within a diaphragmatic hernia. The volvulus may, on the other hand, be entirely intra-abdominal. In contradistinction to the type that occurs within a hernia, such cases present a tremendously distended stomach. Although nausea is constant, attempts to vomit are ineffectual. Only those fluids retained in the esophagus are regurgitated. The obstruction at the esophageal opening is the obvious explanation for this. A barium swallow clearly demonstrates this obstruction.

In those cases in which there is gastric volvulus associated with a diaphragmatic hernia, the symptoms of high intestinal obstruction develop rapidly. Little or no abdominal distention is present and the physical as well as the radiologic findings are those of fluid in the chest. Attempts to pass a gastroduodenal tube are unsuccessful because the obstruction is at the cardiac end of the stomach. This should suggest volvulus to the attending surgeon, especially when the onset has been acute. The sudden onset of severe upper abdominal pain as

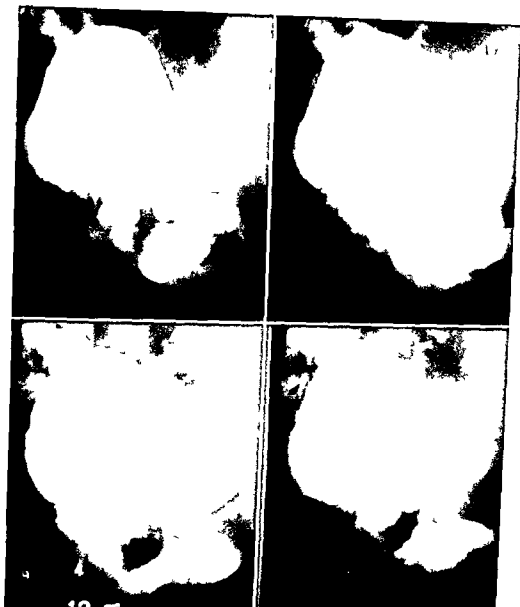


FIG. 37 Volvulus of the stomach. Note the position of the esophagus and the marked displacement of the fundic portion of the stomach.

sociated with an inability to vomit gastric contents is highly suggestive of volvulus. A survey film of the chest and abdomen utilizing air contrast or a barium swallow is confirmatory. Immediate surgical relief of the obstructing volvulus is imperative because of the well known tendency of the stomach to undergo strangulation in such cases.

Isolated cases of gastric volvulus have been reported since Berti's report in 1866. The first surgically treated patient was reported by Berg in 1895. Varying types of volvulus have been reported. The process may involve the entire stomach or only a portion of it. The stomach may ro-

tate on its longitudinal axis or it may rotate at right angles to its long axis. The signs and symptoms produced depend upon the degree of closure of its ends and upon the presence or absence of strangulation. Mild and recurrent volvulus has been described but in no sense does this resemble the acute dramatic onset of a complete volvulus.

Associated pathologic conditions have been reported such as carcinoma of the stomach, gastric ulcer, benign tumors, elongated gastrophagic and gastrocolic omenta, and hiatus hernia. In some cases the splenic flexure and transverse colon are found high in the abdomen. A descriptive classi-

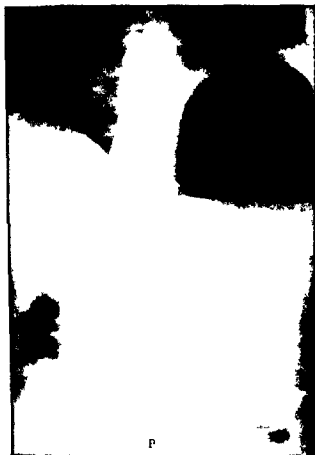


FIG. 28 Volvulus of the stomach. Note tremendous gastric distention and fluid level.



FIG. 39 Volvulus of the stomach which is obstructed by the pylorus as well as at the fundus. Note the tremendous gastric distention.

classification of volvulus of the stomach has been proposed by Singleton as follows:

- A Axis of rotation
 - 1 Organ-axial rotation occurs on relatively fixed points of esophageal and duodenal openings
 - 2 Mesenteric-axial rotation on axis of mesenteric attachments of the stomach
- B Degree of rotation
 - 1 Complete if torsion is 180 degrees or more
 - 2 Incomplete less than 180 degrees
- C Direction anterior or posterior
- D Etiology
 - 1 Associated or contributory pathology
 - 2 Idiopathic if volvulus occurs without evidence of other pathology
- F Acute or chronic

The radiologic findings most commonly noted with gastric volvulus are (1) high position of stomach with cascade appearance (2) narrowing of the pars media with demonstration of spiral

appearance of rugae (3) appearance of the greater curvature of the stomach above the lesser curvature (4) the duodenal bulb pointing downward and to the right and (5) double fluid level.

The treatment of the milder cases may be medical consisting of aspiration of the stomach fluid by the Levin tube and replacement of water and electrolytes. By this regime many of the milder types of volvulus may be spontaneously reduced. However the recurrence rate is high and these cases then require surgical treatment.

Surgery is indicated as the treatment of choice for the recurrent type of chronic volvulus and also for all cases of acute volvulus with complete obstruction. The various type of treatment proposed in the management of these cases range from simple detorsion with correction of associated pathology to sub-total gastrectomy. Decom-



FIG. 40 Note the point of twisting of the stomach. The fundic air bubble is readily seen. Note the point of twisting of the stomach just below this. This film was made after partial correction of the volvulus permitting the barium to pass through.



FIG. 41 Volvulus of the stomach. The twist in this case was on the long axis of the stomach.

pression of the tremendously distended stomach may be necessary before detorsion can be effective. Those cases associated with hiatus hernia require the repair of the hernia as a necessary part of the treatment. Anterior gastropexy has been suggested as the method applicable to those cases which show elongation of the omentum.

INTUSSUSCEPTION

This is the result of peristaltic action upon a pedunculated tumor within the stomach. Chiari in 1888 recorded the first case of this type in which a polyp in the stomach was found to have intussuscepted into the duodenum. Since then reports of a similar nature have appeared in the literature with increasing frequency. The first successfully treated case which is credited to Wade involved a patient in whom a pedunculated myoma of the stomach was found to have caused intermittent attacks of gastric obstruction as it passed through

the pylorus into the duodenum. Ultimately an intussusception occurred as a result of downward peristaltic activity of the duodenum upon the tumor.

Although simple prolapse of the intral mucosa through the pylorus with or without a pedunculated gastric polyp is quite common, actual intussusception of all coats of the stomach is extremely rare. (Amendola reported a case of intussusception of a marked degree in which all coats of the stomach prolapsed through the pylorus obstructing it.) Gastric resection is the procedure of choice in the management of all lesions of this type. Gastro-enterostomy may be utilized in the poor risk or aged patient.

OBSTRUCTION ASSOCIATED WITH DIAPHRAGMATIC HERNIA

Hiatus hernia is a rather common type of diaphragmatic hernia producing such signs and symptoms as pyramidal ulceration and bleeding. In approximately 75 per cent of hiatus hernia cases the treatment is medical. On some occasions incarceration and strangulation of the herniated

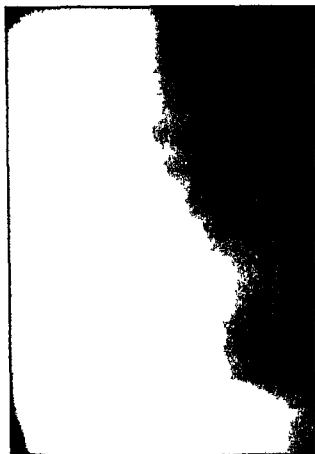


FIG. 42 Hiatus hernia with strangulation of the stomach. Note the two fluid levels.

portion of the stomach may occur. This constitutes a surgical emergency. Perforation of the incarcerated stomach due to impaired blood supply occurs rather early. This must be kept in mind in any case in which a hiatus hernia is suspected or known to be present. A survey film may disclose a greatly distended stomach and fluid levels above and below the diaphragm. Surgical intervention is imperative in all such cases. A Levin tube should be passed into the stomach and the air evacuated prior to surgery. In cases in which the strangulated portion of the stomach is completely closed off, it may be impossible to decompress the stomach below the diaphragm by the Levin tube. The proximally placed portion of the stomach should be emptied nevertheless. Great gentleness in handling the edematous friable stomach is essential if rupture and gross soiling are to be avoided. Pickhardt suggests phrenic nerve interruption as temporary



FIG. 43 Hiatus hernia with strangulation of the stomach.

measure for cases in such poor condition that definitive surgery is impossible. In this case it may be a life saving procedure.

ELECTROLYTE LOSSES

Drinking of water in the presence of pyloric obstruction has been shown experimentally to result in a marked increase in the loss of water and chloride. Marked dechlorination and alkalosis occurred only when obstructed animals were allowed to drink water or when obstructed animals not drinking water were given large volumes of glucose intravenously.

In obstructed experimental animals drinking water, antrum resection was found to prolong life, reduce chloride and water loss in the stomach, and protect against alkalosis in a striking fashion. Vagus section was found to have a similar effect. It has been suggested that water in itself is a mild stimulus to gastric secretion. This may be the re-



FIG 44 Hypertrophic pyloric stenosis in the adult. Notice the "string sign" of the pyloric canal. The persistence of this narrowing is indicative of pyloric stenosis.

sult of antral distention resulting from ingested water.

The creation of a gastric fistula in these experimental animals was shown by Clarke and co-workers to diminish the chloride loss. In the e cases, both the chloride loss and water loss were reduced by antrum resection. The presence of large volumes of hypotonic fluid within the stomach in association with pyloric obstruction has been considered as causing a passive diffusion of electrolytes from the relatively hypertonic extracellular fluid into the gastric lumen. As a result, water drinking increases the water and chloride loss markedly. Drinking of physiologic saline causes no such effect.

HYPERTROPHIC PYLORIC STENOSIS IN THE ADULT

The earliest description of this entity is credited to Cruveilhier in 1837. An autopsy report of 31 adults with pyloric hypertrophy was reported by Muer in 1885. Kirklin and Harris, in a report from the Mayo Clinic, described 81 cases. Only one of these was diagnosed prior to surgery. Isolated cases were reported in the 20 years following the report of Kirklin.



FIG. 45. Note the persistence of the "string sign" in the serial films.

It is generally agreed that the essential pathology is a hypertrophy of the pyloric musculature particularly the circular muscle. This may be so great that it occludes the lumen of the stomach as well as forming the pathognomonic tumor. There is considerable controversy over the etiology of this entity. Two varieties of adult hypertrophic pyloric stenosis have been described. One type presupposes the presence of a mild hypertrophic pyloric stenosis in infancy which becomes accentuated in adult life. The other is a persistent infantile cases. The second variety is believed to be the result of pylorospasm with a resultant muscle hypertrophy. The cause of such spasm may be a peptic ulcerative or bilious tract disease.

Although certain criteria have been proposed to differentiate hypertrophic pyloric stenosis from carcinoma or sarcoma of the antrum, it is generally agreed that an exact diagnosis may be difficult or at times impossible. An exploratory operation may be required before exact diagnosis can be made. Even with the abdomen open a correct diagnosis may not always be easy.

If the findings are classic and a correct diagnosis can be made, the Fredet-Ramstedt operation should be performed. If the diagnosis is in doubt the stomach should be opened and a frozen section made. The presence of malignancy or stenosis



FIG. 46 Microphotograph of hypertrophied muscularis in an adult with hypertrophic pyloric stenosis

ing ulcer necessitates a subtotal gastrectomy. Pylorotomy may be used in an occasional case. Gastroenterostomy without biopsy and frozen section should never be done.

OBSTRUCTION OF THE DUODENUM

Duodenal obstructions may be acute or chronic complete or incomplete. They are caused by a wide variety of mechanisms. Lacking a mesentery and being fixed retroperitoneally the duodenum is immune to some of the obstructive mechanisms found in the small intestine such as volvulus. However because of its retroperitoneal position and peculiar relationship with the pancreas the duodenum is vulnerable to obstruction by compression.

The earliest articles describing chronic obstruction of the duodenum appeared in 1752. Following this few articles of importance appeared until the years 1820 and 1829. The causes for the duodenal obstruction were ascribed to adhesions and compressions.

ADHESIONS

Many papers have been written describing adhesions as a cause of chronic duodenal obstruction. Inflammatory reactions are not held to be an essential cause for all the adhesions around the duodenum many of which are believed to be congenital in origin. Those inflammatory reactions causing obstruction are considered to be secondary to other inflammatory processes in the stomach the gall bladder the cystic duct the liver the transverse mesocolon the ascending colon or the posterior parietal peritoneum. Duodenal ulcerations have been cited as a not uncommon cause for the adhesions. Such adhesions may produce complete or incomplete duodenal obstruction. At times these periduodenal adhesions may be found with no other visceral lesion apparent. Harris described six cases of duodenal obstruction due to a persistence of the anterior mesogastrum as a distinct

fold extending across the ventral surface of the duodenum to the hepatic flexure. This fold then becomes lost in the peritoneum over the pancreas or in the superior surface of the transverse mesocolon. Congenital bands producing obstruction of the duodenojejunal junction are not uncommon and have been described by many authors.

The occurrence of veils membrane or adhesions and bands in the right upper quadrant of the abdomen has been known for some time. Anatomically such bands may represent the unabsorbed portion of the free edge of the lesser omentum and are called the hepatoduodenal, hepatocolic or cystocolic ligament depending upon the actual course of the anomalous membrane. They either cross or attach to the duodenum in its first or second portion. When the first portion alone is involved the deformity of the duodenal cap may be noted radiologically. Bands crossing the second portion of the duodenum produce a characteristic radiologic appearance. When the patient is examined in the erect anterior position immediately after the stomach has begun to empty its barium meal the second portion of the duodenum appears to be pulled out to the right for varying distances and at varying angles. In some cases a sharp angle results between the fixed upper and free lower portion of the duodenum. This acts as a point of transient obstruction.

Kantor considered most of the bands of the right upper quadrant as being congenital in origin although he recognized that some acquired adhesions might also occur. Harvey found that the development of the cystocolic fold reached a stable

condition sometime prior to birth. Bryant indicated that the incidence of the c-bands in the fetus was almost the same as that in adults. An incidence of 48 per cent for adhesions involving the second portion of the duodenum was reported by Kantor in a series of 1734 radiologically studied patients. A review of the literature reveals that 1 to 30 per cent of all anatomically studied cadavers presented such adhesions or bands. In Kantor's series the bands causing obstruction of the duodenum occurred more commonly in females than in males, 79 per cent as compared to 21 per cent. Anatomists, however, report that the distribution of these bands is approximately equal between the sexes.

In the majority of instances symptoms of partial or complete obstruction of the duodenum appear. In 11 per cent of all cases the patients were asymptomatic.

The cholecystoduodenocolic ligament, a special band occasionally causing duodenal obstructions, requires separate consideration. Harris in 1914 first



FIG 48 The cholecystoduodenocolic ligament cut demonstrating compression of the duodenum underlying it

called attention to this ligament as a cause of obstruction when he reported cures as a result of severing it. In the wake of this report numerous case reports appeared in the literature attributing cures to the surgical removal of the cholecystoduodenocolic ligament. Harris believed that this ligament was an embryologic remnant of the caudal edge of the ventral mesogastrium which remained as a fold of peritoneum during the stage of rotation of the bowel and the formation of the liver. As a result it stretched from the gall bladder across the duodenum to the hepatic flexure of the colon.

Although this ligament has been held responsible for symptoms simulating gall bladder disease, duodenal ulcer, and functional colon distress, few cases are reported to have caused major obstruction. The consensus at present holds that with a few exceptions these ligaments are asymptomatic.

In a case reported by Kelley, the congenital band producing obstruction of the duodenum was very dense, about the size of a little finger, and ran obliquely downward from right to left. Inside there appeared a large blood vessel which Kelley thought was the superior mesenteric artery. Failure to recognize the importance of this vessel and a subsequent clamping and cutting of the obstructing band without regard for the vessel would have



FIG 47 Cholecystoduodenocolic ligament producing obstruction of the duodenum. Note the denseness of this band

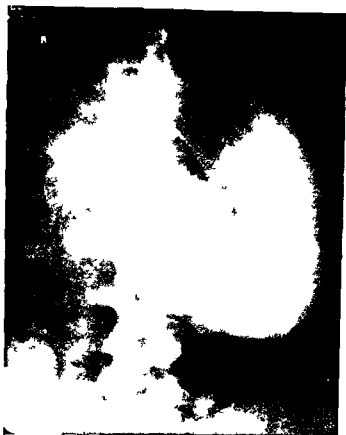


FIG 49 Obstruction of the second limb of the duodenum by recurrent carcinoma following resection of the right colon. Note tremendous gastric distention and retained fluid in the stomach.

resulted in infarction of the small bowel. Exceptions like this make it necessary to consider each case on its own merits.

Gregory, Priest, and Larron reported a case of extreme duodenal obstruction produced by a cholecystoduodenocolic ligament in a 22 year old man. Such complete obstructions are uncommon, however. Harvey reviewed the literature on obstruction caused by this ligament and found an incidence of 17 to 30 per cent reported by authors. He personally reported that the cholecystoduodenocolic ligament was found in 18 per cent of 105 autopsies performed on infants ranging in age from the newly born up to two years.

Samuel has reported an indentation on the greater curvature of the duodenal cap and a deformity of the pyloric antrum as a manifestation of this ligament. Interestingly enough in some cases of this type marked degrees of duodenal obstruction develop within a relatively short period

of time in early adult life without being preceded by upper abdominal discomfort.

ARTERIOMESENERGIC COMPRESSION

The final stage of embryologic rotation of the midgut fixes the third portion of the duodenum between the lumbar portion of the vertebral column and the mesenteric root anteriorly. The mesenteric root contains the superior mesenteric artery, vein, and nerve. It crosses the third portion of the duodenum on its anterior surface. The intermediate position of this portion of the duodenum between the superior mesenteric vessels anteriorly and the vertebral column posteriorly renders it vulnerable to compression. This is apt to occur when for one reason or another the lumbar portion of the spine becomes prominent or the tension of the mesenteric pedicle is too great.

In the quadruped the duodenum is not vulnerable to compression because the drag of the intestine is ventral. In man the drag is caudad in the erect position and ventral only when man is on all fours or in the knee chest position.

Normally the mesentery of the small bowel has a broad posterior attachment and the drag on it is distributed over a broad base. In some cases the mesenteric attachment is small so that the drag of the small intestine is transferred to this narrow mesenteric pedicle. In addition the right side of the colon may lack its posterior fixation so that its weight is added to that of the small intestine. The drag of the small intestine may cause the mesenteric pedicle to become taut across the third portion of the duodenum obstructing it. As a result the duodenum proximal to this point becomes markedly dilated because of chronic obstruction.

Apart from conditions caused by anatomic variations the most common etiologic factor in the production of duodenal occlusion by the mesenteric root is *visceroptosis*. When this occurs the ligaments and mesenteries become over stretched. The small bowel mesentery stretches to such an extent that the loops of small bowel prolapse into the true pelvis. The mesenteric pedicle containing the superior mesenteric artery, vein, and nerve resist overstretching and become taut and band like. The visceroptosis may be associated with



Fig. 50 Duodenal ileus. Fluor. copically barium was retained in the first and second limb of the duodenum for a long period of time.

lumbar lordosis which further predisposes to arterioesenteric compression.

Bockus in a review of the subject reported that Rokitsky in 1849 was the first to suggest that compression of the duodenum by the superior mesenteric vessels might be responsible for dilatation and stasis within the duodenum. Kellogg refers to case reports of duodenal obstruction in 172 by Boener and in 1820 by Yeats. In 1889 Cleland suggested that the drag of the dilated ptosed stomach at times caused a dilatation of the duodenum. Floodgood suggested duodenojejunostomy for the relief of duodenal obstruction in 1907. Strively performed the first successful duodenojejunostomy for chronic gastrosenteric ileus in 1908.

The recent literature makes it apparent that arterioesenteric duodenal ileus (Wilkie's syndrome) is a fairly common disorder. In addition to the congenital factors involved in its causation inflammatory adhesions, tuberculous glands and neoplasms are mentioned as etiologic factors. Tel-

ford reports that in Vancouver one such operation is performed every two months. This would suggest that while the mild forms of such compression are relatively common the severe forms are rather not. This ileus may occur in any age group. It has been reported in 9 week-old infants as well as in 66 year old adults. This latter report by Shrum clearly indicates that age alone does not rule out the presence of this disorder. One of the most unusual reports of this type of compression was that made by Kraffman and Gerbode. In this case a duodenal obstruction was produced by the application of a body cast to a 14 year old boy. This caused compression of the superior mesenteric vessels against the anterior surface of the duodenum obstructing it. Treatment simply involved removal of the cast whereupon the obstructive symptoms promptly disappeared.

In a similar fashion compression of the third portion of the duodenum by the middle colic artery is considered to be due to a low or too loose fixation of the right colon.

As a result of such compression the signs and symptoms of chronic duodenal obstruction appear namely pain, nausea, vomiting and upper abdominal distention. Loss of weight and constipation are not uncommon. Just as in other types of small bowel obstruction the symptoms are seldom all present in any one case. The pain usually comes on after meals particularly heavy meals. It begins directly over the site of the obstruction and radiates proximally along the course of the duodenum. Nausea and vomiting follow the pain. If the site of the obstruction is distal to the ampulla of Vater the vomitus contains bile. The pain and discomfort are usually lessened after vomiting. The degree of epigastric distention depends upon the amount of dilatation of the duodenum and the stomach and also upon the degree of obstruction. The stomach becomes dilated only after obstruction has been present for some time and the pylorus becomes incompetent. Migraine headache is one of the most common toxic symptoms of chronic duodenal obstruction. Loss of weight due to malnutrition because of the obstruction may reach serious proportions.

It must constantly be borne in mind when this lesion is found that such anomalies are often mul-

tuple. A search should be made for others. In the differential diagnosis one must consider disorders of gastric, hepatic, cholecystic, pancreatic or renal origin.

On examination little evidence of diagnostic importance may be present. Some tenderness may be elicited over the point of obstruction. The radiologic examination is of the greatest importance in arriving at a correct diagnosis. Considerable radiologic diagnostic experience is needed to determine whether the duodenum is dilated or not unless the dilatation reaches such proportions that it is obvious even to a neophyte. Nichols believes that the finding of a sizable distention of the second and third portions of the duodenum when correlated with the large duodenal bulb and associated with the characteristic history of duodenal ileus is sufficient to make a correct diagnosis.

The treatment for this type of obstruction may be medical or surgical. The medical treatment involves posture, abdominal support and diet. Patients may discover for themselves that when they assume the knee chest position after meals the discomfort is greatly relieved. A good abdominal support should be tried. Frequent small meals should be eaten. If after a reasonable period no relief is obtained from these methods then surgical intervention must be resorted to. In this event duodenojejunostomy is the procedure of choice. When this is not possible because of the condition of the patient or because of technical difficulties gastrojejunostomy becomes the only other alternative.

ANOMALIES

Atresia of the duodenum may occur generally resulting in death from inanition. Congenital stenosis is not uncommon. It varies greatly in degree from the very high grade obstructions terminating fatally to milder forms of partial stenosis which permit the patient to go on to adult life before symptoms are produced. These atresic and stenotic lesions which were first reported by Calder in 1733 are fully discussed in the chapter dealing with obstruction in infancy and childhood (Chapter 13).

Duodenal diaphragm as a cause of chronic duodenal obstruction has been the subject of voluminous literature since the first report by Poyd in

1845. Just as duodenal stenosis is considered to be caused by the failure of vacuolization of the duodenum after the proliferation of epithelial cells forming a solid core so the faulty vacuolization of this solid core of cells in the duodenum results in the formation of a duodenal diaphragm composed of normal duodenal structures which have failed to reach their proper stage of development. They constitute a possible 2 per cent of all obstructions of the duodenum. Duodenal diaphragms have been reported at all ages and an equal distribution is reported for both sexes. In contrast to cases of stenosis of the duodenum where patients usually do not survive more than four weeks approximately 29 per cent of all duodenal diaphragms were not discovered until the patients were over 24 years of age.

With the exception of some degree of dilatation and hypertrophy, the external appearance of the bowel wall in the region of the diaphragm is normal to inspection and palpation. The degree of dilatation and hypertrophy depends upon the degree of obstruction as well as its duration. The location of the diaphragm in relation to the ampulla of Vater is comparable to that of other intrinsic malformations. It was found proximal to the ampulla in 45 per cent of cases, opposite the ampulla in 20 per cent and distal to it in 25 per cent. The duodenojejunal junction was the site of such diaphragms in only 10 per cent of all cases.

The gross structure of the diaphragm is that of a thin partition extending across the bowel lumen. It may be complete or contain an opening which is variable in size. An occasional variation is the inverted type of lesion which projects down the canal in a finger like fashion. The base of the diaphragm varies generally from 1 to 3 mm. in thickness becoming thin in its central zone.

Microscopically the bowel wall on either side of the lesion appears to be normal. The diaphragm is composed of a continuation of the mucous membrane and submucosa. A few muscle fibers have been found in the base of the diaphragm and in Seidlitz's case islands of pancreatic tissue were found. The relationship of the size of the aperture to the age of the patient and the length of time elapsing before serious symptoms occurred was definite and fairly constant in the younger

reported by Knick. He noted that the size of the aperture did not increase in proportion to the normal lumen of the bowel. Symptoms of obstruction appeared when the hyperperistalsis became marked.

Plumley pointed out that solid foods added to the diet of infant were apt to precipitate acute duodenal obstruction. Faulty mechanical functioning of the bowel wall might result from an incomplete development of muscular and nervous elements associated with the development of the diaphragm in the duodenum.

The symptoms complained of by the patient and the signs noted depend upon the degree of obstruction present as well as dietary and other associated factors. Vomiting is the most frequent symptom and usually the first one to appear. It can be regular or projectile and may occur after every meal. In some cases it appears only at the end of the day. Nausea invariably accompanies the vomiting in patients old enough to complain of it. Pain in the right upper quadrant or umbilical region is present in most cases and may occur at the time of vomiting. Patients find that lying in the prone position affords them some measure of relief. Visible peristalsis has been reported as being observed in only 2 per cent of these cases. The most frequently encountered signs associated with obstruction due to duodenal diaphragm include distention, weakness, dizziness, fever, belching, tumor mass and sudden death.

Rotational anomalies at the time of fetal development may set the stage for the subsequent development of intestinal obstruction. Spencer reported a case in an infant of obstruction of the duodenum which resulted from a failure of intestinal rotation. As a result the common mesentery of the small bowel and right colon produced a compression of the third portion of the duodenum. In addition a torsion of this common mesentery had occurred producing a midgut volvulus. At operation in the case reported by Spencer this volvulus was untwisted, exposing another point of obstruction in the upper jejunum produced by a vascularized sheet of peritoneum which had passed over the bowel. Dissecting this sheet free resulted in successful relief of the obstruction in the patient. From this case of Spencer's as well as from

numerous others, two important lessons can be learned. First, the only way in which volvulus of the small bowel can be properly reduced is by completely excising the small bowel to expose the exact anatomy present. Early surgical intervention is imperative in such cases to avoid the hazards of distention. Second, whenever one anomaly is found causing intestinal obstruction, a diligent search should be made for others. The well known tendency of congenital anomalies to be multiple should constantly be remembered. A further discussion of this subject appears in Chapter 13.

Brown and Ross, in reviewing obstruction due to rotational anomalies, note that such congenital abnormalities of rotation and mesenteric attachment are common causes of intestinal obstruction in the adult. Two cases of mesenterium commune are reported by these authors. In this type of case there are no fixed retroperitoneal third and fourth portions of the duodenum. The duodenum continues from the second portion remaining posterior but mobile on a mesentery which is common to it, the entire small bowel and the right side of the colon. This represents not only a deficiency in intestinal rotation but also the failure of the mesentery to establish its normal attachments to the posterior abdominal wall. Mesenterium commune may also be combined with a failure of intestinal rotation. In cases of this type the second portion of the duodenum is in an anterior position and to the right of the common mesentery. The small bowel occupies the right side of the abdomen and the terminal ileum enters the cecum from right to left. The colon in such cases is found to the left of the common mesentery with its artery.

Right paraduodenal hernia may occur as a result of intestinal rotational abnormality. This is thought to be the result of the duodenum and small bowel being slow in rotating when compared with the right colon. As a result the small bowel becomes trapped beneath the mesentery of the right colon in its process of fixation to the posterior peritoneum.

Among the rather uncommon types of obstruction resulting from rotational disturbances is the case reported by Morlock and Gray. In this patient the second and third portions of the duodenum had invaginated between the leaves of the

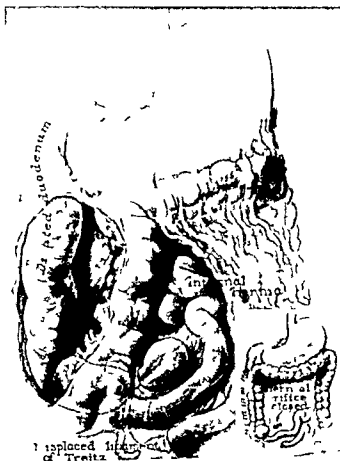


FIG 51 Invagination of the second and third portions of the duodenum between the leaves of the ascending mesocolon. There is incomplete rotation of the colon. Obstruction of the duodenum has resulted from herniation of the first portion of the jejunum through a defect in the mesentery of the ileum.

ascending mesocolon. There was incomplete rotation of the colon. The obstruction of the duodenum resulted from a herniation of the first portion of the jejunum through a defect in the mesentery of the ileum. Here again we find a multiplicity of congenital anomalies eventually causing obstruction.

The developmental anomaly described as annular pancreas occasionally becomes of clinical importance requiring surgical intervention. Obstruction of the second limb of the duodenum with a tremendous proximal distention of the first limb of the duodenum and stomach may result. Annular pancreas is a developmental disturbance in which a firm ring of grossly normal pancreas is found to completely encircle the descending portion of the duodenum. It is continuous with the head of the pancreas without demarcation. In some



FIG 52 Note isthmus of pancreas (I I) causing duodenal obstruction. L liver retracted showing gall bladder GB gall bladder Tr C transverse colon retracted D duodenum I lobes of pancreas after reflection of peritoneum for exposure of duodenum.

cases the ring may not be complete. A three-quarter ring encircling the duodenum may occur such as in the case reported by Johner. There are varying degrees of completeness of the pancreatic ring. As a result varying degrees of obstruction occur. This anomaly frequently produces no symptoms; the presence of the annular pancreas being an incidental autopsy finding or a finding at operation for some other unrelated condition.

In those cases in which annular pancreas does produce symptoms the signs and symptoms are those of high intestinal obstruction; the degree depending upon the amount of compression by the encircling pancreatic ring which partially or completely constricts the second limb of the duodenum. Generally marked dilatation of the first portion of the duodenum and stomach are found. Associated with this lesion other lesions such as benign gastric ulcer, chronic interstitial pancreatitis, hemorrhagic pancreatitis, duodenal ulcer or obstructive jaundice have been reported at one

in one or another. The diagnosis in these cases must be made by the radiologist. Almost invariably partial or complete duodenal obstruction of the second portion of the duodenum can be radiologically demonstrated. However, in some cases the proximal duodenum may be so tremendously dilated that it may be interpreted to be the dilated pyloric end of the stomach. In most of the cases reported a true diagnosis of this specific type of obstruction was not made radiologically. The fact that the duodenum was obstructed was noted and the following possibilities were entertained: (1) stenosing duodenal ulcer, (2) adhesions, (3) malignancy, (4) aberrant vessels, (5) duodenal diaphragm, (6) atresia, (7) possible annular pancreas, (8) definite annular pancreas, (9) unknown causes.

The management of this type of lesion is always surgical. Section of the encircling duodenal ring may be the procedure employed. If so it must be remembered that the ring contains a duct which communicates with the main pancreatic ductal system. For this reason the duct must be carefully ligated at both ends of the resected ring. Failure to do this will result in a pancreatic leakage. As a substitute for resection of the pancreatic ring a duodenal gastrojejunostomy has been found to be a safe, simple and yet effective form of treatment.

EXTRAMURAL COMPRESSION

Enlargements of adjacent organs may occasionally cause duodenal obstruction. Many examples of this kind of obstruction have been reported involving various organs. Enlargement of the spleen may occasionally result in compression of the duodenum obstructing it. The third portion of the duodenum is most commonly involved in this case. Baird and Kirklin reported a case of extraduodenal compression caused by the pressure of an abdominal aortic aneurysm. Anders reviewed 262 cases of duodenal obstruction, 29 of which he found to be caused by compression in one form or another. Since then (1912) the accumulated literature on this subject has been voluminous. Sherwin and Rocke report a case of obstruction of the duodenum caused by chronic pyelonephritis and hydronephrosis. The duodenal obstruction was the result of compression by the

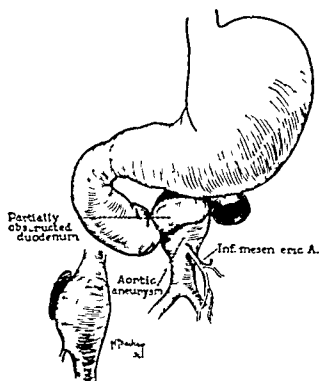


FIG. 3 Obstruction of the third limb of the duodenum by compression of aortic aneurysm.

greatly enlarged right kidney. Removal of the hydronephrotic kidney completely released the duodenal obstruction in this case. When one considers that the second portion of the duodenum and the junction of the second and third portions of the duodenum lie in front of and fixed to the right kidney, the reason for obstruction due to compression by renal pathology becomes obvious.

A rather uncommon type of duodenal obstruction caused by enlargement of the liver due to echinococcus cysts has been reported. Carcinoma of the liver also has been known to produce this type of obstruction. The intimate relationship of the pancreas to the duodenum often results in abnormalities of the pancreas producing obstruction. Carcinoma of the head of the pancreas not uncommonly produces duodenal obstruction. Occasionally carcinoma of the body of the pancreas invades the duodenum at its distal portion obstructing it. Carcinoma of the hepatic flexure of the colon may extend subadjacently to involve and obstruct the duodenum at the junction of its second and third portions. Recurrent carcinoma at the site of ileocolostomy following right colectomy

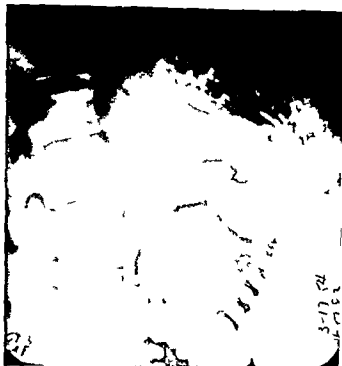


FIG 54 Duodenal obstruction due to chronic interstitial pancreatitis. Note the compression of the second limb of the duodenum by the enlarged pancreatic head.

for carcinoma of the ascending colon is not uncommon. Tremendous gastric and duodenal dilatation may occur because of the slowly progressive nature of the obstructing process.

McMillan and Mison reported an unusual type of duodenal obstruction resulting from a pseudocyst of the pancreas. No etiology could be found to explain the cyst which caused duodenal obstruction by compression.

In 1906 Ochsner described an arrangement of muscle fibers 2 to 4 cm below the ampulla of Vater which he believed might interfere with the passage of duodenal contents as a result of irritation. Inflammatory lesions of the gall bladder were considered to be the most common source for such irritation.

Mesenteric lymph nodes in the region of the duodenojejunal flexure may produce obstruction at this point. Such lymphadenopathy may be inflammatory, tuberculous or due to metastatic malignancy.

FOREIGN BODIES

Foreign bodies such as ingested food or trichobezoars are not uncommon causes for duodenal obstruction. As a rule, however, such foreign

bodies do not lodge in the duodenum. I have reported three cases of duodenal obstruction from ingested foreign bodies. Perry and Shaw found a hair ball lodged at the duodenojejunal flexure obstructing it. Large gallstones occasionally erode into the duodenum and may become impacted in the second portion or at the duodenojejunal flexure producing partial or complete obstruction. Many such cases have been reported.

DIVERTICULA

Duodenal diverticula are generally asymptomatic. The incidence of diverticula of the duodenum has been reported as being approximately 0.8 per cent. They are most commonly found after the age of 50 and appear more often in females. The solitary diverticulum accounts for 90 per cent of all diverticula found. The most common site of origin appears to be the second portion of the duodenum. The solitary diverticulum never appears in the first portion of the duodenum. Although duodenal diverticula as such are generally asymptomatic, their complications are the same as diverticula anywhere in the body. Obstruction, although uncommon, has been reported often enough to merit attention. Here the symptoms associated with obstruction differ little from other obstructive processes except that this variety of obstruction most often follows an inflammatory process in the pouch. Perforation with local abscess formation may occur causing ileus as well as mechanical obstruction from pressure. Treatment is generally conservative. Intubation with gastroduodenal decompression, intravenous alimentation and antibiotic therapy usually adequately correct the disorder. An occasional case may require gastrojejunostomy as a diversionary procedure but complications of this type are rare.

INFECTIONS

Acute pancreatitis may be associated with a duodenal ileus. This may be in part the effect of the generalized ileus from the acute inflammatory process and in part compression of the duodenum by the markedly swollen indurated pancreas. Perforation of a posterior wall ulcer into the lesser peritoneal cavity may produce local peritonitis causing duodenal ileus. The duodenum may be part

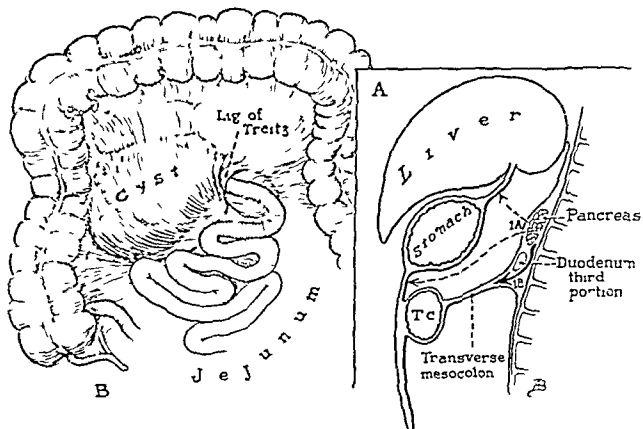


FIG 56 Pancreatic cyst causing duodenal obstruction



FIG 58 Large gallstone in duodenum. Note the relation between duodenum and gall bladder



FIG 57 Note large radiopaque gallstone in duodenum



FIG 58 Duodenal obstruction due to large gallstone. Note retention of barium in the lower end of stomach and marked gastric dilatation. Note also barium in biliary radicals. This is the same patient as that shown in Figure 57.

of the ileus often associated with poliomyelitis. Tuberculosis has been reported as a rare cause for duodenal obstruction. The management of obstructions from this source is purely conservative in involving antibiotic therapy and gastroduodenal decompression.

NEOPLASMS

Benign tumors of the duodenum occasionally become large enough to produce chronic obstructive symptoms. Henderson and Balfour reported benign duodenal tumors in six cases with definite obstruction in only one. The most common benign lesion of the duodenum is the leiomyoma. Approximately 40 per cent of these tumors are found in the jejunum and ileum with the remaining 20 per cent reported in the duodenum. Considering the duodenum alone adenomas are more common than the leiomyomas. Angiomas causing obstruction are rare.

Leiomyomas are generally found in the first

portion of the duodenum with only three cases reported in the second portion. Although some of these tumors are asymptomatic they generally make their presence known. The most common symptoms are vague upper abdominal distress and a feeling of fullness. An occasional case may hemorrhage due to ulceration of the overlying mucosa. The majority of these tumors do not project into the bowel lumen. As a result bowel obstruction is not a prominent feature of this disease.

Carcinoma of the duodenum producing obstruction is not as rare as was formerly thought. Recent reviews of the literature indicate that carcinoma of the duodenum constitutes between 30 and 45 per cent of all small bowel malignancies producing obstruction. Duodenal carcinoma producing obstruction has been reported in slightly over 500 patients which accounts for only 0.3 per cent of all gastrointestinal malignancies. In a comprehensive survey of the literature Teger found that primary carcinoma of the duodenum occurred in 0.033 per cent of 350,000 autopsies. Because of its



FIG 59 Infiltrating tumor of the postbulbar portion of duodenum. Note of obstruction at this point.

ize, however, it has been estimated that much more than one-third of the duodenum is more likely to undergo malignant change than the jejunum or ileum. According to Teger's survey, only 12 per cent of the recorded cases of carcinoma of the duodenum arose in the third portion. In the remaining instances it generally arose close to the duodenojejunal flexure. From this it would appear that the third portion of the duodenum is involved by primary carcinoma least commonly but is commonly obstructed by metastatic carcinoma from the pancreas, stomach or colon.

Duodenal carcinomas producing obstruction are classified according to site of origin as suprapapillary, peripapillary or infrapapillary, the ampulla of Vater being the central point. Peripapillary carcinomas are by far the most common. The suprapapillary are slightly more frequent than the distal duodenal lesions. In addition, carcinoma of the common duct may obstruct the duodenum.

Histologically, primary duodenal carcinomas may be distinguished by their cylindric or spindle cell type and by the actual demonstration of their point of origin in the duodenal mucosa. Although the majority of these lesions begin in the duodenal mucosa, carcinoma has been reported as arising in Brunner's glands, aberrant pancreatic tissue and aberrant gastric tissue. Grossly, these tumors may be polypoid or infiltrating, and tend to encircle the bowel. Metastasis is a late manifestation, occurring in the regional lymph nodes before involvement of the liver or lungs. Intestinal obstruction is usually a late manifestation so that when it occurs, curative resection is often out of the question.

Carcinoma of the duodenum is said to be more common in women than men in a ratio of three to one, with an average age of 50 to 55 years reported. However, it can and does occur below the age of 20. Ungar reviewed 12 cases of carcinoma of the duodenum in patients under 20.

The signs and symptoms associated with carcinoma obstructing the duodenum depend upon the location of the new growth. Generally the tumors grow slowly so that encroachment upon the duodenal lumen is gradual. Carcinoma at the duodenojejunal flexure, however, may produce rather acute constriction. Generally the lesion is far ad-

vanced at the time the patient comes to surgery. The best prognosis can be made when the tumors are found at the duodenojejunal flexure with remote obstruction. Primary resection and anastomosis have been reported to be successful at this site by Pochet and Luquet. Similar good results are being reported in the more recent literature.

The chief symptom of duodenal obstruction due to carcinoma is recurrent vomiting. The vomitus is profuse and may or may not contain bile depending upon whether the obstruction is proximal or distal to the ampulla of Vater. In suprapapillary obstruction, which must be differentiated from a stenosing ulcer, no bile will be found in the vomitus, which will consist only of food. Obstructions at the ampulla of Vater usually contain no bile and may or may not be associated with jaundice. Carcinoma in the infrapapillary portion of the duodenum is characterized by vomiting of both food and bile. This variety of obstruction results in rapid deterioration of the patient because of the loss of pancreatic secretions to the body. The experimental studies of Flman and McCaughan and Flman and Hartmann, as well as Johnstone, Clasen and Orr, have all demonstrated that obstructions below the ampulla of Vater with a loss of pancreatic secretion result in a rapid loss of life in experimental animals. In cases where the obstruction is complete, death of the experimental animal occurred in six to eight days. A considerable loss of weight is invariably an accompaniment of this disorder. In addition to the vomiting and loss of weight, there is nausea and a sensation of epigastric distention which precedes the vomiting and is relieved by it. Because these patients present high intestinal obstruction, they become rapidly dehydrated. Generally there are no abnormal findings on physical examination.

The diagnosis is essentially a radiologic procedure. It has been repeatedly pointed out that obstructions of the third portion of the duodenum may not be demonstrable radiologically. Some radiologists recommend that all radiologic studies be carried out when the symptoms are maximal if an obstructing carcinoma of the third portion of the duodenum is suspected. At times, even when the clinical signs and symptoms of high intestinal obstruction are present, the radiologic examination

may be disappointing in failing to demonstrate the point of obstruction. This is especially true of lesions of the third portion of the duodenum. Lesions at the ampulla and proximal to it lend themselves much better to diagnostic study. Rogers, Golliher and Williams reported negative findings in a patient with carcinoma obstructing the third portion of the duodenum. The suggestion was made that one of the reasons for this lay in the fact that the lesion occurred in the portion of duodenum covered by coils of small bowel or the greater curvature of the stomach and may therefore have been effectively hidden by these overlying barium filled structures. Rogers and Golliher and Williams along with other radiologists have suggested conducting the radiologic examination deliberately during the phase of vomiting. Using this technic a correct diagnosis is possible. Gage used this technic successfully and illustrated it well. It was observed that there often occurred a rapid alternation of severe obstruction with periods of symptomatic quiescence in which the radiologic features were nonobstructive. Golliher proposed four possible explanations for this phenomenon: (1) impaction of solid material at the stenotic site, (2) the occurrence of intussusception at the site of growth, (3) local spasm and (4) varying edema of the growth and overlying mucosa causing obliteration of an already narrowed lumen. The latter explanation appeared to be the most logical for the variation of obstructive symptoms.

Radical pancreaticoduodenectomy is the treatment indicated for ampullary or near ampullary carcinomas. The results are generally disappointing. Segmental resection and end to end anastomosis may be used in those cases in which the tumor is remote from the ampulla of Vater.

NEUROMUSCULAR DISORDERS

Simon and Brown report obstruction of the duodenum with radiologic demonstration of a markedly dilated stomach and duodenum but without evidence of organic obstruction. It is postulated that these cases may be the result of some form of neuromuscular derangement thought to result from a depressive reflex innervation arising in some inflammatory or irritative lesion in another part of the abdomen. Barlow attempted to

show experimentally that incomplete obstruction of the distal portion of the ileum may give rise to dilatation of the duodenum on a neuromuscular basis.

ULCERATION

Duodenal ulceration is probably responsible for the greatest numbers of cases of duodenal obstruction. This most commonly involves the first limb of the duodenum although an occasional ulcerative process may be found below the ampulla of Vater. As a result of the healing process stenosis of the duodenum occurs causing obstruction. This generally occurs as a slow process in a patient who has been subjected to repeated exacerbations and remissions of duodenal ulcer. In some instances "kissing ulcers" concurrent anterior and posterior wall ulcers in apposition to each other may be the cause of duodenal obstruction. The edema in such cases may be such as to completely obstruct the duodenum.

Gastric resection is the procedure of choice in the management of stenosing duodenal ulcer. This is particularly true of those cases in which massive bleeding has occurred from the posterior wall ulcer. In an occasional case primarily that of an aged patient with stenosis of long standing and



FIG. 60. Duodenal ulcer with obstruction. This is the most common cause of duodenal obstruction.

low gastric acidity gastrojejunostomy may be the operative procedure of choice. Under such circumstances a minimal operative procedure is indicated. In the younger patient with normal or high acidity an occasional case will require gastrojejunostomy with vagotomy instead of subtotal gastrectomy whenever there is reasonable doubt that an adequate stump closure is technically pos-

sible. An alternative but somewhat more risky method is the catheter method proposed by Welch for use in the poor stump patient. Essentially the technic consists of performing a subtotal gastrectomy and gastrojejunostomy then closing the duodenal stump around a catheter placed into it and brought out by a stab wound on the side. In addition these cases should all be drained.

OBSTRUCTION OF THE SMALL INTESTINE

There are few abdominal conditions which test the diagnostic ability and surgical judgement of the surgeon to the degree that intestinal obstruction does. Moreover it is one of the most serious conditions arising within the abdomen and unless recognized early and properly treated it carries a high mortality rate.

Intestinal obstruction is essentially a condition in which there is a cessation of the normal forward motion of the intestinal contents for a sufficient period of time to cause local and general changes in the body physiology. It is not necessary that the lumen of the bowel be completely obstructed or that peristaltic activity be completely inactivated or that circulation to the bowel be wholly lacking. While any or all of these three factors may be involved the one factor common to all types of intestinal obstruction is stasis of the intestinal contents.

It has been customary to speak of obstruction of the small bowel as simple or strangulating, depending upon whether the blood supply to the bowel has been compromised. There are many who believe that the so called simple obstruction at times may be treated by intubation alone. (Numerous cases of intestinal obstruction due to adhesions have been grouped under the simple obstruction classification.) In contrast to simple obstruction where a period of delay or study is common while attempts at intestinal decompression using a long intestinal decompression tube are made, strangulating obstruction requires immediate surgery.

However on the basis of experience gained in treating a relatively large number of patients we firmly believe that the simple obstruction theory

is untenable and even dangerous. The term simple obstruction often leads the uninitiated to believe that an obstruction due to adhesive bands without involvement of the circulation is simple indeed. Consequently several days or even a week may be taken to pass a long intestinal tube in such cases. Our experience as well as that of an increasing number of surgeons has shown that it is often impossible for even the most expert surgeon with vast experience and great diagnostic acumen to differentiate in all cases between the simple obstruction and the strangulating obstruction. There are many cases in which all the clinical features presented by the patient are those of simple obstruction and yet at the time of surgery gangrenous bowel is found. For this reason we believe that the term simple obstruction should be deleted from the medical literature. In fact one should consider all obstructions from the surgical point of view as either strangulating or non strangulating. One may then divide intestinal obstruction into many sub groups depending upon the etiologic agent involved in its production. By adopting the viewpoint that all obstructions are potentially dangerous and that there is no such thing as a simple obstruction much of the delay which was typical heretofore in the management of the cases can be avoided.

Numerous authors have stressed the fact that delay in seeking competent surgical attention is the most important single factor in the high mortality rate associated with the management of intestinal obstruction. The longer the period of time between the onset of symptoms and the institution of definitive treatment by the surgeon the lower

the mortality rate will be. An accurate diagnosis of the specific type of intestinal obstruction and the prompt institution of adequate therapy are the *sine qua non* for a low mortality rate. We believe that all cases of mechanical intestinal obstruction regardless of cause should be treated surgically as soon as the condition of the patient permits. On the other hand non mechanical intestinal obstruction such as either paralytic or dynamic ileus should be treated without surgery utilizing the long intestinal decompression tube restoring electrolytes correcting blood volume deficits and using antibiotics in those cases induced by infection. In such cases the ileus is treated as an adjunct to the management of the specific precipitating mechanism.

A tremendous advance in the management of intestinal obstruction was made in 1931 when Wangensteen reported the successful decompression of three acute mechanical intestinal obstructions by continuous suction applied to an in dwelling tube. The mortality rate prior to the introduction of Wangensteen's method of intestinal decompression was appalling. The conservative decompression management of obstruction of the bowel introduced by Wangensteen, brought a rapid and remarkable decrease in the mortality rate. But as is often the case when new concepts are introduced the pendulum swung too far in the direction of conservatism. As a result many patients died who otherwise might have lived had surgical treatment been instituted promptly. Many a case of strangulating obstruction incorrectly diagnosed was treated by intestinal decompression alone only to have the patient die of perforation or gangrene of the obstructed bowel. Recognizing the danger of the indiscriminate use of intestinal suction in the treatment of intestinal obstruction Wangensteen stated in 1939 that the practice of employing suction as a test procedure to indicate whether operation will be necessary leads only to deferment of appropriate treatment. Failure to heed this admonition against the application of suction to the obstructed gastro intestinal tract is one of the most serious errors which can be made. Wangensteen called attention to the fact that suction in such instances was worse than the old method of blind enterostomy.

Small bowel obstruction may be produced by a wide variety of different pathologic conditions which ultimately bring about the same results. It is important that an accurate diagnosis be made as to whether the obstruction is strangulating or non strangulating. To a lesser extent an exact etiologic diagnosis is also important but it is of greater importance to know whether the obstruction is complete or incomplete than to know the specific etiologic factor producing it. In the presence of an acute intestinal obstruction on a mechanical basis from whatever cause prompt surgical intervention is essential if a low mortality rate is to be attained. However a case of incomplete or nonstrangulating obstruction (even one of high degree) accompanied by intestinal distention allows the surgeon adequate time to study the patient and make an exact diagnosis. During this period decompression by long tube intubation and the correction of electrolyte imbalances put the patient in the best condition possible for surgery. This is of course the ideal management for all cases of mechanical intestinal obstruction regardless of cause.

The most serious error in the management of all types of small bowel obstruction is the failure to recognize the presence of a strangulating obstruction. Wangensteen believed that for patients with so-called simple obstruction due to adhesions who were not decompressed satisfactorily by suction decompression enterostomy might still be the operation of choice although many of these patients made uneventful recoveries when treated by intestinal intubation and decompression alone. However a mortality rate of 17.9 per cent in a series of 136 patients reported by Wangensteen and his associates in 1939 gives some indication of the seriousness of simple obstruction.

In reviewing the literature on acute intestinal obstruction it becomes quite apparent that the wide variation in the etiologic factors producing the obstruction depends both upon the medical center reporting and the era in which the report appeared. In a series of 166 cases of intestinal obstruction reported from the Mayo Clinic during the year 1938-1939 the most common single cause of simple obstruction was found to be carcinoma of the sigmoid or rectosigmoid colon. Postoperative adhesions occupied second place. (It is in-

interesting to note that in the same report the mortality rate for all cases of so called simple obstruction was 21.8 per cent.) In other medical centers hernia constituted the largest single cause of intestinal obstruction. At a metropolitan hospital like Grace Hospital in Detroit it was found that by far the largest number of cases of intestinal obstruction was caused by adhesions. The only possible explanation for these variations lies in the fact that different types of hospitals treat patients of different kinds. One would hardly expect the same amount of emergency surgery to be performed at a university or a clinic hospital as at an emergency or a metropolitan hospital in a large industrial city. Therefore the statistics on the etiologic factors producing obstruction must be evaluated on the basis of the center from which they came.

The etiologic factors responsible for acute small bowel obstruction have shown a decided change in the past 50 years. In the material presented by Gibson for the 10 year period from 1888-1898 the following etiologic factors were reported:

Factors	Cases
Hernia	374 (35.4%)
Intussusception	187 (18.7%)
Bands	156 (18.6%)
Volvulus	121 (12.1%)
Meckel's diverticulum	42 (4.2%)
Gallstones	40 (4%)
Openings	34 (3.4%)
Foreign bodies	16 (1.6%)
Miscellaneous	20 (2%)

In comparison the etiologic factors responsible for acute bowel obstruction in a series of 425 patients treated at Grace Hospital 50 years later are as follows:

Factors	Cases
Adhesions	240 (56%)
Hernia	33 (7.7%)
Volvulus	11 (2.5%)
Abdominal abscess	12 (2.8%)
Gallstones	7 (1.6%)
Undetermined	26 (5.9%)
Miscellaneous	96 (21.4%)

(In this latter series disorders responsible for less than five cases each were placed in the miscel-

laneous group. This group included stenosing ileocolitis, atresia of the bowel, internal hernia, Meckel's diverticulum, obstruction at the site of previous anastomosis, tuberculosis, endometriosis, sarcoidosis, lymphangioma of the ileum, ileitis, foreign body, intussusception, anomalies, omentitis, and diverticulitis of the jejunum.)

In comparing these two tables of etiologic factors, 50 years apart, some of the differences are readily explainable. For example, adhesive bands were responsible for the greatest number of obstructions in the Grace Hospital series (56 per cent) whereas in the 1888-1898 series only 18.6 per cent of all cases were obstructed because of adhesions. The vast majority of these adhesions in the recent series were acquired as a result of previous surgery. The tremendous increase in the amount of abdominal surgery performed in the past 20 years as compared with the period from 1888 to 1898 was undoubtedly the causative factor. For instance, the ratio of women to men operated upon is now almost 3:1. This is chiefly the result of the large number of operations performed upon the female generative organs. At the time of Gibson's review in 1898 the proportion of males to females was 1:1 largely because of the relative infrequency with which operations upon the female generative organs were performed. This helps to explain why adhesions as a cause of intestinal obstruction were responsible for only 18.6 per cent of all cases of acute intestinal obstruction at that time as compared with 56 per cent of all cases of obstruction found at Grace Hospital in 1930.

Hernia which was the most common cause of intestinal obstruction in Gibson's series (35.4 per cent) ranked a poor second (7.7 per cent) in 1930. The great reduction in the number of obstructed hernias is due to the high percentage of repair in this era.

Intussusception was responsible for the second largest number of cases obstructed in Gibson's series whereas less than five such cases appeared in the Grace Hospital series. It is difficult to understand why the incidence of intussusception was so high in 1900 and so low in 1930 unless dietary changes played a part.

Volvulus appeared to be five times as common

in 1900 as in 190. That dietary improvements are probably partly responsible for the reduction in the number of these cases is suggested by the fact that in countries such as India where the diet is very meager the incidence of volvulus is much higher than in our country.

Gallstones were the cause of acute small bowel obstruction in 4 per cent of all cases in 1898 but in only 1.6 per cent in 190. Here cholecystectomy for the removal of gallstones has undoubtedly been responsible for the decrease in the number of patients obstructed from this cause. Cholecystectomy at present is a routine and safe operative procedure. In 1898 it was a rather formidable procedure with an appreciable mortality rate and as a result was much less frequently performed.

Figure 61 schematically presents the age grouping of the patients treated for acute small bowel obstruction during the period of the Grace Hospital study. Upon examination it may be noted that the largest group represented in this series of cases occurred in the 20-year period between the ages of 40 and 60. A breakdown of the specific number of patients in each 10-year period is as follows: from age 1 to 10, 6 patients; 10 to 20,

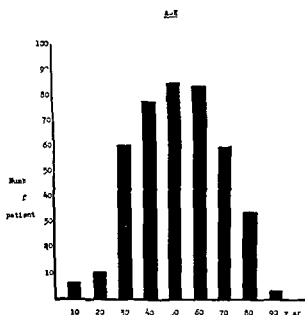


FIG 61 The incidence of small bowel obstruction in the various age groups. Note that the highest incidence of small bowel obstruction occurs in the 20-year period between 40 and 60 years of age.

years, 61 patients; 30 to 40 years, 79 patients; 40 to 50 years, 86 patients; 50 to 60 years, 84 patients; 60 to 70 years, 60 patients; 70 to 80 years, 33 patients; and 80 to 90 years, 3 patients.

In an effort to determine the surgical procedure that most commonly was followed by acute small bowel obstruction the following table was compiled from the 425 patients with obstruction treated at Grace Hospital:

Appendectomy	127 patients
Hysterectomy	66 patients
Bowel obstruction	55 patients
a. Lysis of adhesions	40 patients
b. Resection of bowel	5 patients
Salpingectomy or salpingo-oophorectomy	40 patients
Oophorectomy	33 patients
Cholecystectomy	27 patients
Suspension of uterus	18 patients

Surgical procedures numbering 10 cases or less include resection of the colon, duodenal diverticulectomy, ectopic pregnancy, caesarean section, plication of uterosacral ligaments, dilatation and

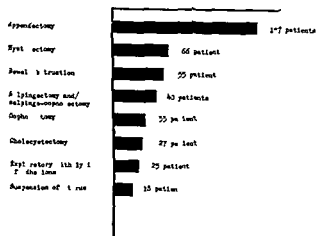


FIG 62 A breakdown of previous surgical procedures contributing to the development of intestinal obstruction. Surgical procedures numbering 10 cases or less: (a) resection of colon; (b) duodenal diverticulectomy; (c) ectopic pregnancy; (d) caesarean section; (e) plication of uterosacral ligaments; (f) c and d with perforation of uterus; (g) gastro-enterostomy; (h) small bowel resection; (i) intussusception; (j) surgery of the bladder; (k) cecostomy; (l) closure of perforated ulcer; (m) ileocolotomy; (n) Meckel's diverticulectomy; (o) ruptured viscus; (p) Noble's plication.

curetage with perforation of uterus gastroenterostomy small bowel resection intussusception surgery of the bladder cecostomy closure of perforated ulcer ileocolostomy Meckel's diverticulectomy and ruptured viscus

DIAGNOSIS

A correct diagnosis of small bowel obstruction including the site and possible cause of the obstruction is desirable if the proper treatment is to be given. Patients with possible intestinal obstruction generally fall into one of three groups.

1 This group consists of all those patients in whom a diagnosis of intestinal obstruction is certain—for example patients with incarcerated hernia in whom the site of blockage is obvious if looked for. Obstructions of the rectum may appear within the range of the examining finger or within the range of the sigmoidoscope. In such cases the sigmoidoscopic examination not only provides a diagnosis of bowel obstruction but also permits biopsy so that a specific diagnosis is possible.

2 In this group of cases the diagnosis of intestinal obstruction is probable and the physician can infer its presence because of the signs and symptoms which the patient presents. Here we find those patients who have been operated upon or have had peritonitis at one time or another and suddenly suffer severe cramping abdominal pain associated with borborygmus and vomiting—probably produced by an adhesive band. Or a child may be admitted with severe abdominal pain and present a mass in the abdomen with a history of bloody diarrhea suggestive of intussusception. Patients in the old age group admitted with a history of alternating constipation and diarrhea, loss of weight and presenting a distended abdomen, anuria and a history of flattened feces should suggest that a stenosing carcinoma of the colon is present.

3 In this group are all those patients in whom the symptoms are vague and the signs indefinite. Here it may be difficult to make a correct diagnosis and one may not even suspect that a bowel obstruction is present. This group taxes the diagnostic acumen of the surgeon and internist and many diagnostic studies may be necessary. One

must not depend too much upon a classic history or the pathognomonic findings commonly associated with intestinal obstruction—pain, vomiting, obstipation, and abdominal distention. In the time all the above findings have appeared the mortality rate will be high because a correct diagnosis will have been made too late.

SIGNS AND SYMPTOMS OF INTESTINAL OBSTRUCTION

Very few of the early cases and only some of the late ones present all the classic signs and symptoms of intestinal obstruction. Those cases in which all the characteristic signs and symptoms have appeared have been referred to as cases presenting the signs of lost opportunity. In the management of intestinal obstruction suspicion alone should be sufficient to justify a presumptive diagnosis and therapy should be instituted at once. The signs and symptoms which are commonly found in intestinal obstruction are as follows:

Pain and Sound

The pain experienced by a patient with intestinal obstruction is colicky in character. It is the result of a stretching of the bowel wall. An increased stretch results from the arrival of each new peristaltic wave causing the characteristic cramp so commonly noted. These cramps come with a regular frequency every four to six minutes. There may be no pain between cramps particularly when the obstruction is nonstrangulating. The cramping pain in intestinal obstruction is associated with borborygmus. This association of pain and sound is one of the characteristic features of bowel obstruction. At the height of the intermittent colicky pain one may hear a gurgling or tinkling sound which has been described as obstructive borborygmus. The noisy abdomen found in mechanical intestinal obstruction is in sharp contrast to the silent abdomen associated with paralytic ileus. Although intestinal intra-abdominal sounds may be audible in a patient suffering from other gastrointestinal disturbances or even from a food allergy, the sound in such case does not occur in exact association with the bouts of cramping pain. The characteristics of early obstruction differ greatly from those of a late obstruction.

even when both are due to the same etiologic factor. Since unrelieved intestinal obstruction gradually develops into an atonic state with an absence of peristaltic sounds the colicky pain with the gurgling, bubbling, or tinkling sounds audible in the abdomen early in the course of intestinal obstruction may not be present later in the course of the disease when the bowel has become atonic.

Intestinal peristaltic activity is manifested by the quantity and the quality of intestinal bowel sounds as easily noted by abdominal auscultation. By auscultation normal peristalsis is a high pitched delicate tinkling sound occurring at intervals and best heard when the patient breathes quietly. In bowel obstruction the intestinal sounds increase in strength and frequency and are associated with colic. The sounds of bowel obstruction arise from the increased action of the proximal bowel as it attempts to overcome the obstruction.

The pain associated with acute small bowel obstruction may be produced in several ways. The greatest stimulus capable of producing pain in intestinal obstruction is an increase in the intraluminal tension as a result of intestinal distention. The more sudden the onset of intestinal distention the greater is the degree of pain. Distention is a well known stimulus to peristaltic activity. As a result an abnormally stretched small bowel responds physiologically with vigorous contractions which are then followed by a period of relaxation. The vigorous contractions cause the intraluminal pressure to increase suddenly. The contractile response is greater than it would be if the same tension were applied to a nondistended bowel so that there is an increase in the pain sensation experienced by the patient. In addition to the pain produced in this fashion any change in the circulation to the bowel wall produces a tissue anoxia which is capable of causing violent contractions of the viable muscle of the bowel wall. These vigorous contractions cause an additional increase in intraluminal pressure and again an increase in pain. In these strangulating cases the severity and acuity of the pain depend upon the speed of onset of the circulatory disturbance within the bowel. Mesenteric thrombosis may cause pain of such severity and stimulation of the bowel to such a degree that the patient may go into irreversible

shock. In those cases in which the circulatory compromise is gradual in onset the pain may be so slight that it is almost unnoticed by the patient.

Nausea and Vomiting

Nausea and vomiting are common findings in intestinal obstruction and are the result of the accumulation of gas and fluid above the point of obstruction. They vary according to the site of obstruction appearing early in high obstructions and late in low obstructions. In obstructions of the colon nausea and vomiting may not be prominent features at any time.

When associated with high intestinal obstruction nausea and vomiting may be of greater importance in determining the outcome than is the obstruction itself. The intestinal stream blockage is serious but not nearly as serious as the disturbances in water and electrolyte metabolism caused by uncontrolled vomiting. The loss of body fluids associated with high intestinal obstruction results in marked dehydration. The loss of sodium and chloride ions can so disturb the electrolyte balance that death results.

The vomitus becomes fecal and foul smelling only in very late cases of low intestinal obstruction. Although some attribute this to a reversed peristalsis similar to that which has been proved to occur in the duodenum the consensus is that the vomiting in low obstruction is due to a reversal of the intestinal stream. This is the result of a normally directed peristaltic wave forcing the intestinal contents against the obstruction so that the stream reverses itself and passes proximally.

Distention

The degree of distention is extremely variable depending upon the site of the obstruction and its completeness. Distention may be entirely absent in high intestinal obstruction. Chronic obstruction of the duodenum on the other hand may be accompanied by an enormous dilatation of the stomach. Generally the degree of abdominal distention increases with the descent in the level of the obstruction. The greatest degrees of abdominal distention occur in obstructions of the rectosigmoid associated with an incompetent ileocecal valve.

Obstipation

Failure to pass gas and feces is not an absolutely reliable criterion of complete bowel obstruction. The ability of the bowel distal to the point of obstruction to empty itself has been established. After the bowel distal to the point of obstruction has been completely emptied nothing further can be expected to be passed with the possible exception of some bloody mucus in cases of carcinoma of the colon. Insisting upon the presence of obstipation for a diagnosis of intestinal obstruction results only in failure to recognize many such obstructions. In fact diarrhea may at times be a prominent feature of obstruction. This is the result of irritation of the rectosigmoid by adjacent inflammatory processes. Since the contents of the small bowel are liquid hyperperistalsis can force small amounts through an almost complete obstruction so that a liquid stool may then be passed. This is apt to give the unwary a false sense of security particularly when it follows surgery. In such cases the differential diagnosis rests between a paralytic ileus and a mechanical obstruction. A considerable amount of time may be lost by treating a patient for paralytic ileus when the obstruction is mechanical in nature.

Wangensteen and Gochl demonstrated experimentally that following complete obstruction the administration of an enema invariably resulted in an evacuation of the distal portion of the bowel. This indicates that bowel evacuation either occurring spontaneously or when induced by an enema does not eliminate intestinal obstruction as a cause of the symptoms. It also indicates that the gastrointestinal function distal to the point of obstruction is normal and responds to stimuli in a normal physiologic manner.

NON STRANGULATING VERSUS STRANGULATING OBSTRUCTIONS

In this past decade it has been taught that intestinal decompression should be tried first in all cases of simple nonstrangulating obstructions. The patient is then observed for a period of time with the expectation that many cases may thus avoid surgery. Proponents of this method feel that those patients who will require surgery later can be spotted early enough to avoid serious damage. We

believe this to be a very unsound practice and deem it dangerous one.

Many cases of intestinal obstruction may be classified as strangulating or nonstrangulating on the basis of the history and physical finding. We do not believe however that there are any criteria by means of which one can tell with absolute certainty when a given case becomes strangulating or if it is strangulating from the very beginning. For this reason we believe that when diagnosis of mechanical small bowel obstruction has been made surgery should be performed as soon as the patient can be put in a satisfactory condition. This may require intravenous alimentation, blood and decompression of the upper gastrointestinal tract. It must be emphasized that we advocate this approach only in cases of acute mechanical obstruction.

Errors in the diagnosis of strangulating obstruction arise for many reasons. The most common of these is the failure to recognize that any obstruction is present. The next most common error is the fact that there is no single pathognomonic sign by means of which the strangulated loop can be identified with absolute certainty. A number of acute abdominal lesions may be confused with strangulating obstructed bowel. A correct interpretation of all available clinical data is essential if an accurate diagnosis is to be reached.

A strangulating obstruction is defined as one in which there is an interference with the blood supply to the segment of bowel in addition to the blockage of the intestinal stream. The extent and character of the circulatory impairment is dictated with the strangulating obstruction as well as the symptoms associated with this catastrophe depend upon the site at which the obstruction to the mesenteric blood supply occurred and the speed with which the obstructing process took place. When the vascular obstruction is close to the origin of the principal mesenteric vessel the area of bowel which is impaired is large. The closer the obstruction is to the mesenteric edge of the small bowel the less involvement there will be. Thus a true because of the rich anastomatic blood supply in the small bowel wall. A single thrombus obstruction of the superior mesenteric artery will cause an infarction of the entire small intestine as well as part of

the right colon. However a single thrombus in one of the lesser arteries close to the bowel may cause no detectable change. A vascular obstruction of sudden onset which completely blocks the blood supply causes more immediate and severe effects upon the bowel than does one in which the onset is more gradual and less complete. In the slowly occurring type of vascular obstruction the decreased blood supply through the occluded vascular channels may to a variable extent be compensated for by the development of a collateral circulation.

A reduction in the blood supply to the bowel wall below the level necessary for the maintenance of normal function causes a tissue anoxia. Under the stimulus of this anoxia the smooth muscle of the bowel increases the force and frequency of its contractions. As a result hyperperistalsis occurs. A persistence of the vascular impairment causes the bowel to lose its power of contractile response. It may become spastic then flaccid and finally paralytic. A continuation of the vascular obstruction results in necrosis of the bowel. This then perforates producing peritonitis.

Perforation is not necessary before bacteria from within the bowel lumen can reach the peritoneal cavity. There is ample proof that organisms capable of causing peritoneal irritation or inflammation can gain access to the peritoneal cavity through the non necrotic bowel wall. This occurs because of the increased permeability of the bowel due to long standing anoxia.

Richardson called attention to the rusty blood stained fluid found in the peritoneal cavity in cases of strangulation of the bowel. He suggested that the appearance of such peritoneal fluid might aid in the diagnosis of intestinal obstruction after the abdomen had been opened. This observation by Richardson in 1920 was the result of his study of 135 cases of intestinal obstruction 22 of which were strangulated and contained this typical fluid. The same observation has been made repeatedly by subsequent surgeons. Hill, O'Loughlin and Stoner demonstrated in an experimental study on dogs that peritoneal aspiration in the diagnosis of strangulated bowel was feasible. This finding has been applied to humans by many surgeons in recent years.

It may be difficult to diagnose strangulation in

patients seen for the first time. Some of the features that have been found to be helpful in reaching a diagnosis of strangulation are:

1. An increase in the amount of pain which is noted while under observation. This is a valuable symptom. The pain becomes much more severe with the onset of strangulation.

2. Development of abdominal tenderness. If present previously it increases along with the increase in severity of the pain.

3. There is an increase in the pulse rate with the onset of strangulation.

4. Development of an abdominal mass. This suggests the presence of a strangulated loop of bowel which has become distended.

5. Muscle spasm may make its appearance. This is indicative of peritoneal irritation.

6. Perforation of the obstructed loop may cause peritonitis with its characteristic physical findings.

7. Leukocytosis usually develops fairly early in the process of strangulation. This may be absolute or relative as shown by a shift to the left of immature leukocytes.

8. A sudden drop in blood pressure may occur with the onset of strangulation. A drop in blood pressure in any patient suspected of having a strangulated obstruction suggests a perforation of the bowel. Although this is not diagnostic it is helpful in evaluating the progress of the disease.

Although the above points are helpful in the examination of a patient their absence should not be construed as being clear cut evidence against the presence of a strangulating obstruction. Generally one or more of the above signs and symptoms will be found if carefully looked for although they may vary so in degree that they can be easily overlooked. In some cases none of the so called signs of strangulation are found despite careful examination.

Evans studied the mechanism of shock in intestinal strangulation. He was able to demonstrate that strangulation of a short loop of ileum resulted in a sufficiently large loss of plasma locally to account for the fall in blood volume and blood pressure associated with shock. He found no evidence for plasma loss from capillaries damaged by toxins remote from the site of injury. Adapting

these findings to our present day concept of shock yields the following sequence of events

1 An initial phase In this the loop of bowel becomes strangulated by torsion by a band or by being incarcerated within a hernial sac The venous flow becomes obstructed while the arterial flow is unimpaired The result is a high venous pressure within the loop This causes an increase in capillary filtration locally When the endothelial capillary wall becomes damaged fluid and protein pour out into the lumen of the bowel loop which is strangulated as well as through its wall into the free peritoneal cavity Soon almost pure plasma passes through the capillary wall The net result is a reduction in the circulating plasma volume This falls in direct ratio to the local loss of plasma The final result is a reduction in the effective blood volume

2 The sustaining phase occurs in which the blood pressure remains normal only as long as the plasma loss is compensated for by the vasoconstrictor mechanism of the body At this time the damage is still limited to the affected intestinal loop When the plasma loss exceeds 25 to 30 per cent of the circulating blood volume the vasoconstrictor mechanism ceases to be effective because of the disproportion between the effective blood volume and the size of the vascular bed As a result of this the blood pressure drops and the cardiac output diminishes A persistence of this stage for any prolonged period of time results in a diminution of blood flow to the tissues to an extent that generalized capillary permeability due to anoxia develops This leads to the terminal phase

3 The terminal phase is characterized by generalized capillary damage This is attended by a rapid leakage of protein and fluid from the vascular bed to the extracellular lymph spaces This results in pulmonary edema capillary stasis and necrosis of the bowel wall The bowel wall of the strangulated loop becomes permeable to its contents which diffuse into the peritoneal cavity General toxemia develops resulting in death

A shocklike state may also occur in response to marked stimulation of afferent visceral nerve tracts in the obstructed bowel The abnormal stimulation of the visceral afferent nervous fibers in the mesentery of the involved bowel segment is

apt to result in a very early appearance of a shocklike state The same mechanism may be produced by traction upon the mesentery by the etiologic mechanism responsible for the intestinal obstruction The signs and symptoms of peripheral vascular collapse associated with strangulating obstruction could be explained partially on a reflex basis This would be particularly applicable early in the course of the disease before distention dehydration disturbed electrolyte balance and plasma loss enter into the picture It has been known for some time that blood is poured into the lumen of the bowel in strangulating types of obstruction If the obstructed strangulated loop is a long one this blood loss may be considerable

Aufmann and Method studied the role of vascular spasm in the recovery of strangulated bowel They noted that the recovery of the strangulated intestine depended upon a variety of factors The residual vasospasm in the minute vessels of the bowel of a still viable loop was considered to be of great importance as far as the recoverability of the loop was concerned There appeared to be little difference whether the strangulation was primarily arterial or venous as far as the vasospasm was concerned The gross appearance of a still viable arterial type of strangulation was different from that of a still viable venous type of strangulation By the time an arterial strangulation assumed the gross appearance of a venous type its musculature had lost all reactivity and was beyond recovery An arterial type of strangulation was found to retain its recoverability more than five times as long as the venous type The release of a viable arterial type of strangulation results in a reactive hyperemia which is followed by a phase of vasospasm Papaverine hydrochloride may be of value in releasing this residual vasospasm This aids in the recovery of bowel following strangulation provided the loop is recoverable

ETIOLOGY

The causes of acute small bowel obstruction are numerous Some types are more likely to undergo strangulation than others For example any mechanical small bowel obstruction has a tendency to strangulate The impairment of blood supply in any case of small bowel obstruction is of the great

est importance. In all cases of obstruction there are two main problems: (1) a loss of fluids and electrolytes, and (2) interference with the blood supply to the obstructed bowel. The latter may be due to primary interference with the vessels supplying the bowel or it may be secondary to intestinal distention as a result of compression of the vessels in the bowel wall.

Adhesions

Adhesions have generally been found to be the most important cause of small bowel obstruction, although in some areas they may rank second or even third in importance. In a series of cases reported from Grace Hospital, 6 per cent was due to adhesions. In a series of cases reported by Cole, only 32 per cent was caused by adhesions. In a report from the Mayo Clinic in 1940, adhesions were found to rank second to hernia as a cause of intestinal obstruction, accounting for approximately 16 per cent of all cases. In a report on intestinal obstruction, Lupton noted that two-thirds or more of all mechanical obstruction cases in his series were due to external hernia, bands, and adhesions. While most bands and adhesions were postoperative, it was estimated that from 5 to 15 per cent of all cases occurred with no history of previous surgery. Thus a history of no previous surgery or abdominal trauma and the absence of an abdominal scar indicating previous surgery should in no way be considered as proof that an adhesive band may not be causing intestinal obstruction. Such bands may be congenital in origin or may be acquired shortly after birth or at any time thereafter as the result of a long forgotten trauma.

Appendectomy and pelvic surgery were the surgical procedures most commonly followed by intestinal obstruction. It has been established that any patient who has been operated upon for intestinal obstruction due to adhesions runs a much greater risk of recurrent obstruction from the same cause. It has been estimated that from 7 to 10 per cent of the patients who have been obstructed by adhesions may expect a recurrence. The number of patients developing intestinal obstruction as a result of congenital adhesions has been variously estimated from 3 to 15 per cent of

all cases. However, this latter figure seems to be quite high; a 5 per cent average is more likely.

The number of patients obstructed by adhesions has shown a marked increase in the past 20 years. The previously mentioned incidence of 18.6 per cent reported by Gibson when compared with an average incidence of 10 per cent at the present time at Grace Hospital speaks for itself. It is probable that the percentage will increase even further as a result of the tremendous amount of abdominal surgery being performed at the present time.

Obstruction due to bands and adhesions has been classified by Melver as follows: (1) early postoperative, (2) late postoperative, and (3) those without operation. The incidence of early postoperative obstruction due to adhesions is probably much higher than the surgical statistics would indicate due to the fact that many such cases are treated by intubation alone. This is possible because strangulating obstructions are uncommon during the early postoperative period. It is in cases



FIG. 63 Obstruction of the small bowel following appendectomy in the immediate postoperative period. Note the marked small bowel distention.

of this type that many surgeons feel justified in attempting conservative management. Such management often relieves the obstruction for the time being. The use of the long intestinal decompression tube is most effective in this type of obstruction. In a group of 38 patients of this type treated at Grace Hospital by intubation alone, a follow up showed that 13 of these (26 per cent) were readmitted within one year for acute small bowel obstruction due to adhesions. In the series reported by McIver, appendectomy was found to head the list as the cause for early postoperative obstruction. Bowel obstruction after appendectomy may be produced in several fashions. A late diagnosis of appendicitis with the removal of a ruptured appendix may cause a local abscess to form. A loop of bowel adherent to this may become obstructed. Adhesions formed as a result of the inflammatory process may set the stage for the subsequent development of intestinal obstruction. The paralytic ileus associated with appendiceal abscess is well known. There is little doubt that

the indiscriminate removal of normal appendix has been followed in many instances by intestinal obstruction due to adhesions. It is best to avoid the operation of incidental appendectomy unless the appendix itself presents sufficient gross pathology to justify its removal. The appendiceal stump may act as a nidus causing intussusception of the cecum.

A diagnosis of acute mechanical small bowel obstruction in the early postoperative period is difficult. At this time the laboratory is of no help. The temperature, pulse, and blood count are within normal limits. Cramping abdominal pain or tenderness is apt to be ascribed to postoperative gas pains. Since peristaltic activity is depressed or absent during the first 24 to 48 hours after surgery, auscultation is of little help. The abdomen does not present the loudly audible borborygmi resulting from hyperperistalsis of an acute mechanical bowel obstruction during this early postoperative period.



FIG. 64 Paralytic ileus secondary to inflammatory mass in the right lower quadrant as a result of a ruptured appendix.



FIG. 65 Fecinitis with paralytic ileus. Note the gas tube which is well down the gastrointestinal tract after 48 hours.

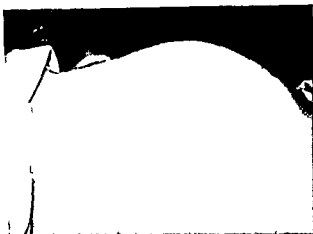


FIG. 66 Note the marked abdominal distention and the right paramedian scar from a cholecystectomy 10 years previously. The presence of this scar might lead one to diagnose intestinal obstruction due to adhesions. However, the small bowel obstruction in this case was caused by iliac compression from a gangrenous appendix.



FIG. 67 Complete small bowel obstruction in a 75 year old woman. The tip of the appendix had become adherent to the mesenteric root and the appendix had obstructed the ileum that it crossed. This is the same patient as the one shown in Figure 66.

Much has been written in the past 20 years to stress the dangers of delay in the diagnosis of intestinal obstruction. A large part of this delay was attributed to the lack of diagnostic acumen of the

general practitioner who first saw the patient. The expression was frequently heard that "Cases of intestinal obstruction first seen by the general practitioner not properly diagnosed stopped for surgery on their way to the grave." The fallacy of such observations is made readily apparent by a review of all statistics which conclusively demonstrate that much of the high mortality rate associated with intestinal obstruction occurs as the result of deaths in the early postoperative period. This sort of obstruction occurs under the very nose of the attending surgeon who fails to diagnose it, attributing the intestinal distention, obstruction, and pain to postoperative ileus or gas pains. There is little doubt that many surgeons not alive to the dangers inherent in delayed diagnosis and treatment place themselves in the unenviable position of operating upon such patients after the bowel has become gangrenous. Such patients are often well along the road to their Maker. In this connection the surgeon must shoulder his share of the responsibility in the management of this type of obstruction with the practitioner who first saw the patient and the internist under whose management the patient may be hospitalized. We must constantly bear in mind the fact that intestinal obstruction is one disease for which operation must at times be performed upon suspicion of obstruction rather than when the diagnosis is established beyond any question of doubt.

Obstruction as a result of adhesions in the late postoperative period is generally a more serious problem because strangulation and volvulus are quite common. In former years resection of an ovary for benign ovarian cysts was often a cause of intestinal obstruction due to adhesions. In these cases the cyst was generally enucleated and the cystic cavity closed or the cyst was punctured leaving a raw area. It was not infrequent in such cases for adhesions to form binding the terminal ileum in an inflammatory mass obstructing it. Suture material at times resulted in intestinal obstruction. Snodgrass reported a rather unusual type of intestinal obstruction caused by the use of nonabsorbable sutures introduced 24 years previously. In this case a loop of bowel had passed through the loop of nonabsorbable suture material and had become obstructed. A review of the literature on

this subject revealed many cases of intestinal obstruction caused by both absorbable and nonabsorbable suture material. In a series of 317 cases of intestinal obstruction reported by Albitsky, 13 were found to have been caused by sutures. It is not unusual to find intestinal obstruction due to leaving long tails on the cut sutures following pelvic surgery and appendectomy. A loop of bowel may become attached to this long tail of suture material and become acutely strangulated producing acute mechanical obstruction or a loop of bowel may become adherent to a suture and form a loop. In such cases a second loop of bowel may slip through the first loop fastened in this way and become strangulated by compression of its mesentery. When a loop of bowel adheres to the abdominal wall uterus or adnexa at one small point permitting a free range of motion of the loop volvulus may occur. The suggestion has been made that the Arthus phenomenon may be provoked in that sensitized individual by cutgut suture which being a sheep protein is an antigen. The reaction to this cutgut has been suggested as a possible source of the inflammatory reaction causing adhesions with adherence of touching structures.

Strangulation is found more frequently in obstructions which occur some time after surgery. In several series of cases appendicitis surgery was found to be the most common cause for the subsequent obstruction. At Grace Hospital pelvic surgery was found to be the most common cause for this late obstruction. Previous surgery with a resultant formation of peritoneal adhesions has been found to be the most frequent of the required factors predisposing to the development of volvulus. The involved loop may be adherent to the abdominal scar, the posterior surface of the uterus or the posterior surface of the broad ligament. The greater omentum may become adherent to the anterior abdominal wall to the posterior surface of the uterus or to the adnexa because of the common surgical practice of placing the omentum well down in the pelvis. As a result a loop of small bowel may twist around this bridge of omentum and become obstructed. In adult small bowel volvulus is usually the direct result of postoperative or inflammatory adhesions and in a much smaller number of cases is due to congenital bands

Among the uncommon causes of intestinal obstruction due to adhesions are those formed by calcified mesenteric tuberculous glands. These may be present for a great number of years and may either be asymptomatic or produce intermittent attacks of partial obstruction from the very onset. The diagnosis in these cases may be made by the radiologic finding of calcified nodes in the region of the umbilicus. In the cases reported intestinal obstruction was produced by adhesive bands from the calcified lymph node or by scar tissue from the calcified lymph node completely obstructing the bowel. The node in itself did not produce the obstruction. The circumferential tissue often reacts to its presence by forming bands between it and the bowel. These bands become the obstructing mechanism.

Cohen and Ichnus report an uncommon variety of obstruction due to appendicitis. In this type the patient may develop an attack of acute appendicitis during which the tip of the appendix becomes attached to the parietal peritoneum the mesentery or to a loop of small bowel. The acute inflammatory process in the appendix then subside and the appendix behaves exactly like an adhesive band. The sling formed by the appendix makes a loop through which a loop of bowel may herniate becoming obstructed. The swelling which occurs in the wall of the trapped loop gradually

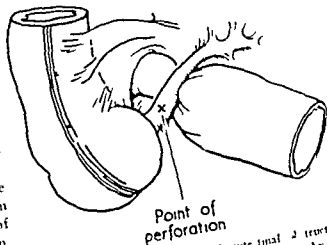


FIG. 29. Acute appendicitis with intestinal obstruction as a result of encasement of the terminal ileum by the acutely inflamed appendix.

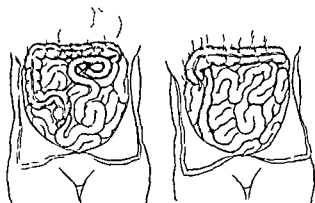


FIG. 69. Small bowel volvulus. Note the anomalies predisposing to the development of this condition. *Left*: Volvulus here included only the small bowel. The cecum has almost gained the right iliac fossa; its mesentery has partly fused with the postparietal peritoneum; fixation was partial. Early and abnormal fusion of the mesentery of the first loop of jejunum with the mesentery of the transverse colon had occurred and an adhesion developed from the cecum across the duodenum at this area. The attachment of the mesentery of the remaining portion of the small bowel was over a comparatively small area. Hence too much mobility and volvulus. *Right*: Early fixation of the cecum preventing its descent to the right iliac fossa.

shuts off the circulation so that strangulation occurs.

Obstruction from bands and adhesions without previous surgery is not common. Approximately 5 per cent of obstructions due to adhesions are ascribed to this source. These bands may be congenital in origin or may be the result of abdominal trauma long since forgotten. Subserosal hemorrhage as well as mesenteric hemorrhage due to trauma may result in the formation of adhesive bands. This is the result of the fibroblastic proliferation associated with the healing process.

One must consider the ligament of Treitz in its broadest sense among the congenital bands causing obstruction. Intestinal obstruction at the duodenojejunal flexure may result from angulation due to a shortened ligament of Treitz at this point. Mullen has reported a case of this type in which tremendous distention of the stomach and duodenum resulted when the partial obstruction caused by the shortened ligament of Treitz was made acute by the weighted distended proximal jejunum loop pulling down upon the duodeno-jejunal flexure.

Hernia

Hernia is a cause of intestinal obstruction ranked a poor second in the series of obstructions reported from Grace Hospital being responsible for only 7.7 per cent. Fifty years ago it ranked first in Gibson's series being responsible for 35.4 per cent of all obstruction. Boyce and McEtridge reviewed a series of obstructions and noted a change in the distribution of cases according to etiologic factors over the past 30 years. In the period from 1923 through 1927 hernia was the most common cause of intestinal obstruction. In a series of cases from 1930 to 1935 hernia still remained the most common cause of intestinal obstruction with adhesions a secondary cause. Following this period they noted that hernia had dropped to second place in importance and that adhesions had become the most important cause of obstruction. In a review from the Mayo Clinic in 1940 hernia (external) ranked first as the most common cause of intestinal obstruction accounting for 30 per cent of all cases. In certain sections of the country, particularly rural communities, the incidence of intestinal obstruction due to hernia is higher than that for any other single cause. In industrial communities where hernial repair is essential in order to be gainfully employed, the incidence of obstruction due to hernia is reduced. This is evidence that prophylactic herniotomy can and will eventually result in a reduction in intestinal obstruction from this cause. Indeed, one would expect that in this modern era of surgery, the efficient methods of hernial repair would already be reflected in a reduction in this obstructive complication throughout the country. However, although the operation of herniotomy is today one of the most commonly performed procedures, the fact still remains that hernia at this time continues to be one of the leading causes of acute intestinal obstruction.

In the treatment of obstructions of the gastrointestinal tract associated with hernia, one must differentiate between incarceration and strangulation. The treatment and prognosis are different for these two conditions. Incarceration may exist for long periods of time without symptoms and hence go untreated, whereas strangulation produces symp-

GASTRO INTESTINAL OBSTRUCTION

agement of obstructive lesions due to hernia on bowel reduction should be severely criticized since nonviable bowel and omentum may be returned to the peritoneal cavity. As a result necrosis of the bowel wall with perforation and peritonitis may occur. Protrusion of omentum and strangulation of adipose tissue in the hernial sac may prevent an accurate picture. It may be impossible to determine whether bowel is present in the sac without surgery. Strangulation in hernia is found to appear more frequently in association with femoral and umbilical hernias than with inguinal. The incidence of femoral and umbilical hernia is less than that of inguinal hernia. In some cases of incarcerated hernia with possible strangulation the hernia may reduce itself following spinal anesthesia. It is important when such spontaneous reductions occur that the contents of the hernial sac be found and examined. Such strangulated bowel may go on to perforation within the abdomen.

Irreducibility is not necessarily synonymous with obstruction. A hernia complicated by strangulation or torsion of omentum may as a result of reflex peritoneal irritation produce a paralytic ileus. Vomiting and pain may then occur in association with it. According to statistics the duration of acute symptoms before treatment is shortest in these cases. Femoral hernia ranks second in importance from the standpoint of frequency but here the duration of acute symptoms before surgical treatment is much longer. Femoral hernia has the highest average age level and the highest mortality rate. In addition femoral hernia is most common in women.

Among the less common obstructive hernias the ventral hernia, the umbilical hernia and the direct inguinal hernia are seen to have equal duration of symptoms before surgery. The mortality rate is almost the same for this entire group. Ventral and umbilical hernias are found most commonly in females whereas inguinal hernias are more common in males.

Among the uncommon types of hernia producing intestinal obstruction are the Spigelian hernia, interstitial hernia and Richter's hernia. This latter does not produce acute intestinal obstruction by interrupting the direct continuity of the bowel. It does however produce a paralytic ileus on a re-

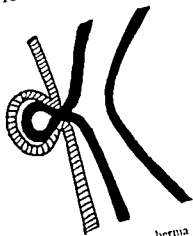


FIG. 70 Diagram of a Richter's hernia in which only a portion of the bowel's lumen is occluded. Note that the continuity of the bowel has not been interfered with.

flex basis. Richter's hernia is particularly apt to perforate and cause peritonitis.

There is an unavoidable mortality rate of approximately 6 per cent associated with the management of strangulated hernia. However this figure is much lower than those quoted prior to the introduction of the antibiotics. The highest mortality rate has been reported for males between the ages of 30 to 79. Although no correlation between the duration of symptoms and the presence of gangrene has been definitely established there is a statistically valid correlation between the duration of symptoms and the mortality. The incidence of strangulation has been reported as being highest in the femoral hernia group. However this has not been our experience. We have found the majority of strangulations in the inguinal hernia group. Most statistical reviews point out that the incidence of males subject to strangulating femoral hernia is twice that of females. Most of the deaths occur when necrosis of the bowel is evident at the time of surgery necessitating bowel resection. Although with the use of antibiotics it would seem that resection of the bowel and anastomosis could now be performed much more safely than heretofore a review of cases reveals that the mortality rate for this type of obstruction is still high. This is particularly true for the series in which the viability of the bowel found within the hernial sac is dubious. But the hope that it will be viable We believe that in doubtful cases when one cannot be certain the bowel within the sac is viable it is

far better to do a primary resection and anastomosis than risk the replacement of nonviable bowel. In the inguinal hernia group the mortality rate increases as the length of segment of strangulated bowel increases. The presence of a grossly bloody or blood tinged fluid within the sac is suggestive of irreversible bowel damage although this is not invariable.

In an experimental study to determine criteria for the viability of strangulated intestinal loops it was found that with experience a considerable degree of accuracy was possible on the basis of the gross characteristics alone. Of these the consistency of the strangulated intestine and the return of color after the release of the obstruction were of the greatest value. Mesenteric pulsations and the amount and character of exudate present were misleading. The absence of pulsations over the mesenteric vessels could not be accepted as evidence of occlusion of these vessels.

Internal Hernias This category encompasses all the protrusions of intra-abdominal contents

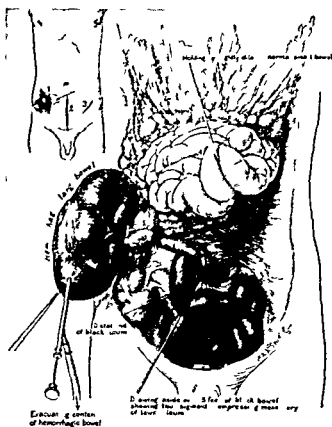


FIG 72 Intestinal obstruction due to a hole in the mesentery of the ascending colon. Note the gangrenous bowel. Note that the strangulated bowel is a descending limb of the colon and a portion of the sigmoid flexure which passed through a dense hernial ring in the mesentery of the ascending colon. Note also small bowel obstruction produced by compression of the circulation of the small bowel passing beneath this arch which resulted in the destruction of more than 5 feet of small bowel to a point within a few centimeters of the ileocecal valve. *Upper left* Diagram showing incisions made during the operation. Incision 1 was a gridiron incision made for the removal of the appendix. Black bowel appeared whereupon the midline incision (2) was made. Incision 3 was made at the end of the operation and the sigmoid attached at that point to be opened later if necessary.

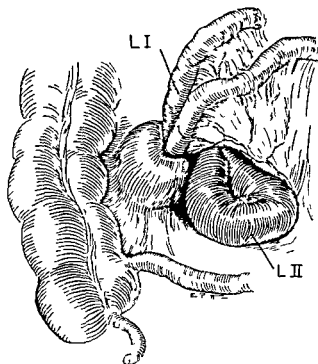


FIG 71 Herniation of the small bowel through a defect in the right mesocolon. Note a secondary point of intestinal obstruction (L II) below the arching mesentery of the first loop.

into intraperitoneal pouches or openings of congenital traumatic or surgical origin. These hernias are relatively uncommon compared with the external varieties. The internal hernia is clinically important because of the high incidence of intestinal obstruction associated with it. There are numerous varieties of these hernias. Among them may be found the retrocolic, the ileoappendicular, the intersigmoid, the retroperitoneal, those through

GASTRO INTESTINAL OBSTRUCTION

the foramen of Winslow through the transverse mesocolon into paraduodenal fossae through rents in the mesentery through openings in broad ligaments through diaphragmatic recesses through the perineum through artificial recesses after colon resections through or around ileostomies as a result of gastro enterostomies or partial gastrectomies and several other rarely encountered varieties.

Paraduodenal Fossa Hernia Landzert first described the anatomy of the paraduodenal fossa in 1871. The subject remained academic until Moynihan established its status as a clinical entity in 1906. He described nine different types of fossa about the duodenum in which a hernia may occur. He reported 74 examples of paraduodenal hernia collected from the literature up to that time.

Not all paraduodenal fossa hernias become symptomatic. Approximately 60 per cent of all such hernias are found accidentally during autopsy or during operative procedure for some other clinical disorder. The clinical group producing acute intestinal obstruction accounts for only 30 per cent of all cases. These were operated upon for signs and symptoms of acute intestinal obstruction of unknown etiology. It would appear that a large percentage of paraduodenal hernias are clinically silent so that the actual incidence cannot be determined. They only become important when their benign course is complicated by some degree of obstruction.

Of the nine duodenal fossae described by Moynihan, the left paraduodenal fossa of Landzert produces 70 per cent of all the duodenal hernial obstructions. This fossa is formed just to the left of the terminal portion of the duodenum on the anterior surface of which two thin nonvascular folds pass laterally to merge with the posterior parietal peritoneum along the line of the inferior mesenteric vein. As a result a superior and inferior crescentic fold are formed. Coursing laterally these become continuous with a false fold of posterior peritoneum raised by the inferior mesenteric vein. A peritoneal pouch is formed to the left of the duodenal jejunal flexure with its orifice to the right and its anterior crescentic neck formed by the peritoneum covering the inferior mesenteric vein. A herniation into this left para-

duodenal fossa of Landzert passes behind the inferior mesenteric vein and presents as a true peritoneal sac in the retroperitoneal region of the left gutter.

Although the pre operative diagnosis of intestinal obstruction due to this hernia is difficult it is not impossible. A typical patient is one with a history of recurrent attacks of indigestion associated with occasional attacks of epigastric pain and suddenly develops vomiting and severe pain in the upper abdomen and presents all the findings of an acute intestinal obstruction. When this patient is found to present a slightly movable and tympanic globular mass localized to the epigastrium a suspicion of a paraduodenal hernia should be aroused. The mass formed may vary in size from that of a grapefruit to that of a bowling ball. Patients with this type of obstruction may complain of rectal bleeding. On examination large hemorrhoidal masses are found in association with obstructed veins are found as a result of compression of the paraduodenal hernia by the hernial sac. Bleeding from the lower colon is not uncommon in such cases due to the back pressure created by the inferior mesenteric venous system by the compression.

The contents of the hernial sac in the paraduodenal hernias vary from a few inches of jejunum to a massive inclusion of the entire small intestine with a portion of the right colon. Obstructions may occur in all types. The larger hernias provide the predisposing factors of a large hernial sac with a comparatively small unyielding vascular margin. This latter facilitates obstruction at the neck of the sac. A mobile cecal mass with fixation of the cecum predisposes to volvulus in addition whereas intrasacculary adhesions lead to simple obstruction.

Retroperitoneal Hernias The cecal hernias may be a variety of the paraduodenal fossa hernia or they may be due to developmental anomalies. The congenital origin of the paraduodenal and other fossae appears to be well documented. The congenital version of the embryonal status of the gastrointestinal tract with its primitive dorsal mesentery into its adult form and the migration of the cecum make abnormalities in the enteric fixation and re-

relationships possible. The fetal type of mesentery with a mobile cecum or incomplete rotation of the colon is not infrequently associated with retroperitoneal hernias.

The symptoms produced by intestinal obstruction due to this type of hernia do not differ from those produced by intestinal obstruction from any other cause. The physical finding of an enlarged, tense, irreducible and painful mass which is constant in position in the epigastrium suggests that a retroperitoneal hernia is present. In these cases the signs and symptoms come on fairly rapidly. Nausea and vomiting are common. Intestinal distention is generally absent because the small bowel involved is so often jejunum. Late in the course of the disease distention may appear.

Mesenteric Rents. Mesenteric rents or holes may be considered infrequent causes of intestinal obstruction. In the 1000 cases of intestinal obstruction reported by Gibson there were only 10 in which the obstruction was the result of herniation of bowel through a hole in the mesentery. Turell reviewed the literature on this subject in 1932. He reported 34 cases including one of his own. In these cases the small intestine was the most commonly strangulated portion of the intestinal tract to herniate through these openings. There were only two cases in which colon herniated through a rent in the mesentery and became obstructed.

These rents or holes may occur anywhere in the mesentery or mesocolon. The most common location is the mesentery near the terminal ileum. These mesenteric holes are slitlike and their edges are smooth, rounded and regular. They are a potential source of internal hernia and obstruction. Treves in his study of mesenteric defects noted that the ileocolic branch of the superior mesenteric artery circumscribes by its anastomosis with the last intestinal artery an area in the mesentery which is well rounded or oval. This area is unusual in that it presents no fat, no visible blood vessels and is never occupied by any mesenteric glands. Cribriform openings may be noted in this area. Aside from this area in the mesentery of the terminal ileum Treves believed that such rents or holes were rare in other parts of the gastrointestinal tract. McIver in a series of 335 cases of obstruction reported two cases in which the ob-

structing mechanism was a mesenteric rent. Many isolated case reports of this type of hernia have appeared in the past 20 years.

Although the aperture is often found proximal to the ileocecal junction it has been reported at any point along the whole length of the mesentery. As a result herniation has been reported in any portion of the primitive dorsal mesentery except the descending mesocolon.

As an explanation of the origin of these holes it has been pointed out that in man there is a regression of only the ventral mesentery whereas in other animals the dorsal mesentery is involved. The congenital rent in the mesentery may thus represent a partial regression of the dorsal mesentery in humans. This would be an atavistic trait. The case reported by Long in which multiple holes were found in the mesentery would appear to support this theory. Another theory about the apertures is the concept that a hole is made in the mesentery by pressure of a loop of bowel at the seventh week of intra uterine life when it is pushed out of the abdominal cavity into the umbilical cord. Iagnov and Timus reviewed the subject extensively and concluded that such mesenteric defects were congenital in origin.

In an occasional case there is a definite history of trauma preceding the onset of symptoms suggesting a traumatic origin for the rent. At operation the opening may be found to be a narrow slit with ragged edges further suggesting traumatic origin. In most cases the opening is round or oval with smooth and firm edges.

In no case reported was a correct pre operative diagnosis made. The greatest difficulty in treatment is to decide whether surgery is indicated. If the abdomen is opened early simple reduction of the hernia and repair of the defect are all that is required. In late cases resection of the bowel may be necessary because such cases strangulate early due to the pressure of the mesentery of the herniated bowel against the firm ring forming the opening of the defect. Strenger reported a case of herniation of 30 cm. of ileum through a defect in the small bowel mesentery 20 cm. from the ileocecal junction. The defect was 6 cm. in diameter. The bowel was completely obstructed but not strangulated.

An unusual variety of this type of defect was reported by Lee. A large foramen was found in the mesentery of the small bowel. A loop consisting of part of the transverse colon, the cecum and the terminal ileum had herniated through this opening. The defect was associated with a reversed rotation of the midgut.

Fenestra of various sizes are not unusual in an otherwise normal omentum. Prolapse of small bowel through such fenestra has been reported. This type of hernia is rather uncommon. Martzloff reviewed the literature on this subject up to 1930 and was able to find only 16 previously reported cases in addition to his own. After this report McI can report another case making a total of 17 cases reported as of 1932.

Holes in Broad Ligament as a Cause of In-



FIG. 73 Note the dense fibrous tissue lining a defect in the omentum. In this patient herniation of the small bowel occurred through this opening with resulting strangulation.

ternal Obstructed Hernia The operation known as Baldy Webster suspension first introduced by Webster and Baldy in the early 1900's was used extensively and successfully for many years to correct retroversion/flexion of the uterus. In 1911 Webster reviewed the technic for this operation calling attention to the closure of the opening in the broad ligament through which the round ligaments are pulled. He emphasized that the holes should be closed by stitching the edges to the round ligament. Despite this warning many such holes were left unclosed. Richardson in 1920 and Pemberton in 1925 reported intestinal obstruction due to herniation of a loop of small bowel through the opening left in the broad ligament and Parkes and Karabin in their review of the literature in 1939 found similar case reports by Suter and Arnold. In a review in 1939 of 100 cases of the Baldy Webster operation performed at the Evanson Hospital Parkes and Karabin noted that not one single operative record mentioned the closure



FIG. 74 Intestinal obstruction caused by herniation of a loop of terminal ileum through a defect in the right broad ligament following Baldy Webster type of suspension. Note the tremendously small bowel distention.



FIG 75 The same patient following surgery. The obstruction was released. Note the remaining marked small bowel distention.

of the opening in the broad ligament suggesting that in each instance this important step in the operation had been omitted.

We have operated upon two patients for acute intestinal obstruction caused by herniation of small bowel through an opening in the broad ligament. Up to 8 inches of small bowel were found to be herniated through such openings. Strangulation is common in such cases.

Rent in Meso Appendix. Combined internal and external hernia with incarceration of the small bowel such as that reported by Thiessen and Rouch is one of the rarest types of obstruction. In this case there was an incarceration of bowel through a congenital defect in the appendical mesentery. A loop of terminal ileum herniated through the causing intestinal obstruction. Associated with this an external inguinal hernia was also present. To release the obstructed bowel it was necessary to sever the meso appendix. No other mesenteric defects were found in this patient. The fact that there was no previous report of a defect

in the appendical mesentery as a cause of intestinal obstruction suggests that such defects must be rare.

Diaphragmatic Hernia. Diaphragmatic hernia due to trauma may cause acute intestinal obstruction. These herniations in contrast to the congenital diaphragmatic hernia present no sac and may be found at any point in the diaphragm. (The congenital diaphragmatic hernia such as the para-hiatal through the foramen of Morgagni or through the foramen of Bochdalek are true hernias and do present a sac. The hernia in such cases is usually of the sliding type.) Traumatic hernias are usually found on the left side because the liver protects the right side of the diaphragm from herniation. These traumatic hernias may contain small bowel, spleen, liver, transverse colon, splenic flexure, stomach or omentum. The variety and length of organs found will depend upon the size and location of the hernia.

A congenital hernia may be asymptomatic or may present vague symptoms for many years and then suddenly become obstructed. The prime consideration in cases of this type is the correction of the obstruction. The question as to whether the hernia should be repaired at the same time depends upon the condition of the patient and the obstructed bowel.

A transabdominal approach is desirable in those cases in which small bowel is noted in the hernia or if the patient is seen late. With this approach not only can decompression of the small bowel be obtained but if the bowel has strangulated, resection and anastomosis can be readily performed. If the patient is seen early and intestinal distention is not an important factor or if the patient shows no evidence of electrolyte imbalance and hydration is good a transthoracic approach is the simplest. The herniated contents in the thorax may be returned to the abdominal cavity and the hernial defect in the diaphragm closed with relative ease. In late cases of acute intestinal obstruction involving the small bowel through a diaphragmatic hernial defect the least surgery to release the obstruction is the best surgery. To accomplish this a midline abdominal incision may be rapidly made. Through this the obstruction can be relieved. The hernial defect cannot be safely or easily repaired. For this

reason a second operation which may be trans-thoracic must be performed to correct the pathology in the diaphragm. Some patients seen late in the course of the obstruction are in such poor condition that the added thoracic exposure may be sufficient to cause death. The treatment of the intestinal obstruction is emergency surgery whereas the correction of the diaphragmatic hernia is elective. In all such cases the treatment of the acute intestinal obstruction is of primary importance.

Intersigmoid Fossa Hernia The small bowel was obstructed in 26 of the 27 cases of intersigmoid hernia reported by Stephenson. In an additional case the sigmoid colon was incarcerated and obstructed in the hernial sac. This is the second case of this type reported.

Perineal Hernia This is one of the rarest forms of internal herniation. This hernia may be classified as anterior or posterior perineal being separated by the transverse perineal muscles. In the female the anterior perineal hernia may be divided into an anterior labial hernia which passes through the pelvic floor and partly encircles the vagina to reach the center of the labia. The posterior perineal hernia descends through the ischio-rectal fossa to reach the posterior portion of the labia. In the male a posterior perineal hernia may occur between the bladder and rectum just lateral to the midline. In the female it may be found between the uterus and the rectum. The usual exit is through an opening in the levator ani muscles or between the levator ani and the coccygeus. In addition to these there is a type of perineal hernia which is caused by a persistence of the embryonic pouch of Douglas which extends down to the levator ani muscle. At birth this pouch becomes elevated to the level of the second or third sacral vertebra. The pouch may become completely obliterated. A partial obliteration of this pouch can be a constricted mouth with a patent sac below. The hernias are median perineal hernias.

Although intestinal obstruction from this source is rare the hernia is an important one because of the high mortality rate associated with it. This is chiefly the result of diagnostic difficulties. MacKenzie, Schwartz and Kolbertazzi have reported a typical case of this type.

Hernia and Obstruction after Gastric and

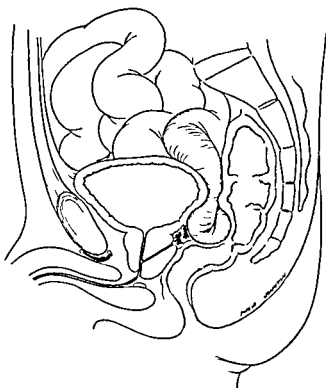


FIG. 76 Strangulation of a loop of small bowel through a hernia in the pouch of Douglas

Colonic Surgery Gastric and colonic surgery may cause pouch and recess formation into which loops of bowel may herniate and become obstructed. These accidents are quite varied in type and are presented in detail in the chapter devoted to these problems (Chapter 24).

Sequelae Following Reduction of Strangulated Hernia As a result of obstruction of the bowel in a hernia necrosis of its wall may occur in one of three different fashions.

1. There may be local pressure necrosis as a result of a tight band or adhesion across a loop of bowel.

2. There may be a constriction of a loop of bowel by the neck of the hernial sac. This is common in a Richter's hernia. In the latter case must be taken because of the ease with which perforation occurs through the necrotic area.

3. Ischemic necrosis may occur in a closed loop obstruction due to the effect of gaseous distention upon the bowel wall. When the pressure within the lumen of the bowel exceeds the capillary pressure within its wall areas of ischemic necrosis may result. These are often found on the antimesenteric border.

After the reduction of an incarcerated loop of bowel in a hernia in which the viability of the bowel was adequate stenotic changes may occur in the damaged areas. The first record of this complication was reported by Gauguier in 1864. Cotte and Iriche collected 39 cases of this type in 1905. Two types of stricture may develop from this cause. A stricture may be physiologic as a result of a temporary interference with the intestinal motility. This produces a spasm of the bowel involved. Strictures of this type disappear when the neuromuscular mechanism is restored to normal. Organic strictures develop gradually and persist until relieved by resection. The organic strictures are of two types: (1) the annular variety which involves a small segment of bowel at either end of the constricted loop and (2) the tubular variety which produces longer constriction and is thought to be the result of impaired blood supply to the entire loop of incarcerated bowel. The intestine proximal to such points of constriction becomes markedly dilated and the muscle layers become hypertrophied as a compensatory mechanism to overcome the obstruction. The bowel below the constriction becomes thin and decreased in size. Many months are required before the organic stenosis reaches a point in development sufficient to produce intestinal obstruction. Warren and Catell reported a case in which eight months elapsed from the time of inguinal hernia repair until surgery for small bowel obstruction due to stenosis.

Because such stenotic areas may develop constricted points in the bowel found in incarcerated hernias should be critically examined. It may be wise to resect such bowel in which the wall induration is high grade to avoid secondary stenosis. *If a watchful waiting policy is elected the patient should be informed of the possibility of developing a stenotic lesion at the point of constriction. He should be urged to seek medical attention without delay at the first sign of gastrointestinal stasis.*

Ingested Foreign Bodies Causing Obstruction. A large number of widely varied foreign bodies have been found in the gastrointestinal tract producing intestinal obstruction as a result of different mechanisms. Among the ingested foreign bodies which have caused obstruction are

knives spoons forks pencils razor blades nails screws combs buttons tooth picks bones of all kinds needles beer mug handles fruit pits fibrous bolus of vegetable fibers from the Japanese persimmon mushroom fiber oats poppy seeds apples prunes fish scales cherry stones gooseberry skins baked fruits dried pears locusts orange fiber and seeds figs suetkraut cherry skins cucumbers potatoes corn kernels and bran. The use of aluminum hydroxide gel administered to old or very ill patients may also result in obstruction. Havens reported a case of intestinal obstruction produced by the indiscriminate use of this gel.

Ingested foreign bodies generally pass through the gastrointestinal tract in five to seven days. Intestinal obstruction as a result of the ingestion of such foreign bodies may be produced by the arrest of the foreign body at the point of narrowing within the gastrointestinal tract. This may cause mucosal ulceration followed by perforation and peritonitis. Paralytic ileus then results. If walling off occurs a localized abscess may result. Intestinal obstruction can then be produced by two mechanisms: (1) pressure of the abscess—adherence of a loop of bowel to an abscess may cause sharp angulation and obstruction or (2) obstruction may be produced simply by virtue of the mass of ingested material. This latter is common with persimmon seeds and has been reported after ingestion of watermelon seeds.

Orange pulp may produce intestinal obstruction. Such patients are generally edentulous and report that shortly after the ingestion of the orange pulp symptoms of the abdominal pain distention and vomiting began. The site of obstruction is generally found to be the terminal ileum.

The size of the foreign body ingested or its sharpness appears to have little influence upon whether it will obstruct. In a series of cases studied by Carp, 83 per cent of all sharp foreign bodies passed without incident through the gastrointestinal tract whereas only 79 per cent of all dull foreign bodies passed without incident. Perforating foreign bodies represented only a small percentage of those ingested. In this group the toothpick was the most common offender. Since no portion of the bowel escapes perforation there is no one site of predilection. Toothpicks may pro-

duce bowel obstruction in one of two ways (1) the bowel may be obstructed as a result of edema which occludes its lumen or (2) the bowel may be completely perforated so that local or general peritonitis results. Waterworth has reported a case of this latter variety. Such foreign bodies do not produce bowel obstruction because of their mass but rather because they may penetrate the bowel wall producing local edema, granuloma of the wall, localized peritonitis, diffuse peritonitis, or abdominal wall abscess. Although ingested foreign bodies often leave the bowel by the seventh day, there are many reports in which they remained within the bowel for weeks, months or even years. Burk, Mann and Karsh reported a fruit pit which remained within the bowel for $4\frac{1}{2}$ years and then began to produce intermittent intestinal obstruction. Such arrests in the passage of foreign bodies may be due to stenosis, adhesions or compressions of the lumen of the bowel. Rambo, Lasky and Iden describe a rather unusual case of acute intestinal obstruction as a result of the swallowing of four turtle eggs. These had passed through the stomach and pylorus to the ileocecal valve where they became lodged. Surgical intervention became necessary. The membranous covering of the eggs was so leathery and tough that it was impossible to break them open even four days after their removal except by cutting them with a sharp instrument. This patient was treated by ileotomy with the removal of the four turtle eggs.

In the Arctic countries and in Africa where ascariid infestation is common, intestinal obstruction due to billing of ascariid worms is not uncommon. Diagnosis of the cause of obstruction becomes fairly certain after the passage of some of the worms per rectum. In one case reported by Ramberg, 500 worms were removed from several areas of the bowel. As many as 1498 worms have been reported in a case of intestinal obstruction due to ascariid. In most cases the obstruction is partial. These patients generally come to surgery after five to seven days of medical management. Distention is not an important diagnostic feature of obstructions from this cause because the ascariid usually inhabit the upper small intestine. In addition, intestinal gases can pass through the tangled mass of worms even though intestinal contents

may be completely blocked. The vomiting associated with ascariid infection may be due to a reflex mechanism or may be the result of a mechanical obstruction. However, only a small percentage of infected patients require surgery because of bowel obstruction.

Among the unusual foreign bodies producing intestinal obstruction, one of the most unusual was that reported by Pfeiffer. In this case a condom firmly tied and distended with ejaculate fluid and air, caused complete intestinal blockage. Weinstein, Roberts and Suss reported a similar case of intestinal obstruction which occurred two months after a condom filled with water was swallowed. This obstruction occurred at the terminal ileum.

Left in the Abdomen. Foreign bodies left within the peritoneal cavity still remain a hazard of abdominal surgery despite all the accepted precautions. One of the earliest cases was that reported by Wilson in 1884, in which a sponge was left in the peritoneal cavity after surgery. (However, general reports of foreign bodies lost within the peritoneal cavity appeared as early as 1646. One report by Fabricius Hildanus concerned a man wounded by a sword thrust into the abdomen. The point of the sword broke off and was lost within the peritoneal cavity where it remained for one year until it was excreted in the stool. Isolated bizarre accounts of this type were reported in the literature of those early times.)

In recent years, similar unusual intraperitoneal foreign bodies have been found. Saltzstein and Rao found a laparotomy towel within the jejunum 10 inches from the duodenojejunal junction. The bowel in this case had become tremendously dilated. How the towel reached this position was unknown. In the course of a routine dissection of a cadaver, Latimer also reported finding a towel which had produced obstruction of the jejunum. The towel producing the obstruction measured 40 by 24 cm. The creases in the towel were still evident after its removal; these creases suggested that it had been folded once longitudinally and twice transversely. The towel was of the type used to drape the body for surgery. In both cases the towel had eroded into the lumen of the bowel obstructing it. Apparently the erosion into the lumen had been so gradual that the bowel had

closed behind the towel and thus leakage and peritonitis did not occur.

In other cases in which foreign bodies were reported lost in the peritoneal cavity many were found excreted in the stool. Only a few were found arrested in the small bowel and in these cases the ileum was involved. In several cases where a sponge had passed into the bowel the bowel presented extensive adhesions indicating severe inflammatory reaction. Fair reported an unusual case of this type as a result of surgery performed 30 years previously. A sterile abscess was found about the disintegrated gauze sponge. The immediate cause of obstruction was the result of the adherent bowel breaking loose from the calcified mass which had been attached to its mesentery so that a hole was made in the mesentery of the small bowel. A major portion of the small bowel had herniated through this opening and become twisted. A volvulus with obstruction resulted.

Another mechanism by which a sponge lost within the peritoneal cavity caused obstruction was noted by Lenthauer and Cantor in 1934. At operation a laparotomy sponge was found with a metal ring attached to it. A loop of bowel had herniated through the metal ring and become obstructed. The ring was so hard that it could not be cut. Resection of the small bowel and end to end anastomosis were necessary to correct the obstruction even though strangulation had not occurred. Crossen and Crossen surveyed the literature in 1940 and found 307 cases of intraperitoneal foreign bodies as the result of surgery reported up to that time. They pointed out that this probably represented only a small fraction of the occurrence of this accident because of the natural reluctance to publicize this sort of case.

Gallstones

Gallstones can cause many types of intestinal obstruction. It is generally believed that gallstones presuppose an internal fistula between the gall bladder or common duct and some portion of the gastrointestinal tract. There have been isolated reports suggesting the possibility that such stones may pass through the common duct and into the duodenum without actual fistula formation.

The first case report of intestinal obstruction caused by a gallstone was made by Bartholin in 1654. Murchison first described the occurrence and frequency of perforation of gallstones into the intestinal tract in 1877. In Gibson's series of intestinal obstruction reported in 1898, gallstone ileus was found in 4 per cent of cases. Walters and Snell reviewed the subject in 1940 and reported an incidence of 2 per cent. Hand and Gilmore summarized a total of 1213 cases of intestinal obstruction reported by 11 publications and found an incidence of 17 per cent for gallstone ileus. We reviewed 425 cases of intestinal obstruction over a 5 year period and found an incidence of 1.6 per cent for gallstone ileus. From this data it appears that the incidence of gallstone ileus has shown a 50 per cent decrease in the past 50 years.

Gallstone ileus occurs more commonly in women since biliary calculi are four times as frequent in women as in men. It is most frequent in the age group beyond 60. Although isolated cases have been reported in younger patients biliary calculi do not usually produce intestinal obstruction in patients under 60. The average age for this disorder is 60 to 70 years.

The mechanism by which the gallstone passes from the biliary tract into the gastrointestinal tract appears to be uniform in most cases although it is varied in a small percentage of instances. The mechanism most commonly responsible for this passage is the erosion of a gallstone from an acutely inflamed gall bladder into the duodenum. The stone may erode into the stomach or into the colon directly. The usual sequence of events is an attack of acute cholecystitis with a large stone in the gall bladder. As the result of the acute inflammatory process gangrene and perforation of the gall bladder occur into the adherent duodenum, colon or small bowel. The formation of a cholecystoenteric fistula and discharge of the gallstone probably take several days. The gallstone may remain protruding into the duodenum or it may pass into the stomach. In some instances the stone erodes through the common bile duct into the duodenum. In any event this erosion may make itself evident by vomiting or by the passage of blood into the gastro-intestinal tract. Bright red

blood may be vomited or passed per rectum. If peristaltic activity is slow, tarry stool will be indicative of this bleeding. The present consensus that the majority of gall stones reach the gastrointestinal tract by means of internal fistula formation is further supported by the occasional appearance of such fistulas into the bladder or kidney. As a result, a gallstone may be found in the urinary bladder or kidney. In a case reported by Elsner, one gallstone was found in the kidney while another had passed down the ureter, through the urethra, and been excreted. A sinus tract was found to lead from the gallbladder to a perinephritic abscess with a secondary kidney perforation.

Perforation of the gallbladder may occur almost asymptotically. As a result, the patient may not be aware of any abnormality until the sudden onset of obstruction. This serves to cloud the diagnosis because there may be no history of an acute episode of intra-abdominal disease. Cases have been reported in which no fistula could be demonstrated between the gall bladder and the gastrointestinal tract. From this, one might suspect that the stones had passed through the com-

mon bile duct. Murphy reported one case in which the common bile duct was so dilated that a stone 4 cm. in diameter could readily have passed through it. Rigler reported the passage of gall stones of large size through the common duct by erosion directly through its wall into the duodenum. In 7 of the 35 cases of gallstone obstruction studied by Courvoisier, the path taken by the stone was through the common bile duct itself. In one of these cases, the common bile duct had dilated to such size that a stone 4 inches in circumference had passed through it and caused bowel obstruction.

Intestinal obstruction as a result of a gallstone may occur at any point along the gastrointestinal tract for any one or all of the three following reasons: (1) because the gallstone itself is too large to pass on; (2) because the gallstone causes intussusception of the bowel; or (3) because the gallstone becomes imbedded in the wall of the bowel, secondarily closing its lumen. A gallstone of a size small enough to pass through the gastro-



Fig. 77 Mr. F. F., age 88, female. Black stool for three months. Pain in all four quadrants. Vomiting. No previous surgery. Note large gallstone producing duodenal obstruction.



Fig. 78 X-ray shadows over the liver. The shadow of the gallstone is visible. Note the large gallstone producing duodenal obstruction.



FIG. 77 Note the duodenocolic fistula

intestinal tract may produce obstruction because of spasm induced within the bowel or because of a partial obstruction of the loop of bowel caused by adhesions, kinking, or angulation. The size and number of stones found within the gastro intestinal tract vary from small stones which pass uninterrupted and are excreted per rectum to those of very large size. It is generally believed that a

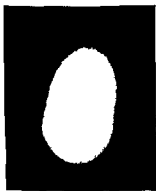


FIG. 80 Gallstone removed from a case of gallstone ileus. Note the radiopaque center of stone and the relative radiolucency of the periphery

stone must be at least 2½ cm. in diameter before it can produce bowel obstruction although smaller stones have been known to obstruct because of the mechanisms described above. A round stone is thought to be more apt to cause trouble than an oval one. It has been estimated that about 25 per cent of all stones present in the gastro intestinal tract producing partial obstruction or even complete obstruction will be excreted per rectum without operative interference.

Turner reported one of the largest gallstones producing intestinal obstruction. The obstruction was caused by two faceted stones in the transverse colon, one stone weighing 4500 grains and measuring 3 by 2½ by 7 inches in circumference while the smaller stone weighed 960 grains and measured 1¼ by 1½ by 4½ inches. Analysis of the large stone showed that it consisted of cholesterol and bilirubin pigments and proved that it was really a giant sized gallstone.

Stones that pass through the ileocecal valve generally are excreted per rectum. However this does not always occur particularly if the pelvic colon is constricted by adhesions. Turner reported one case in which a large calculus obstructed the bowel just above the anus. Among the giant stones producing obstruction the stone described by Burnett deserves mention. It measured 14 cm. in diameter and weighed 636 grains.



FIG. 81 Multiple stones present in the small bowel producing obstruction. (Same patient as in Figure 84)

Two cases have been reported in which the gall bladder perforated into the mesentery of the small bowel forming an abscess. The resultant inflammatory mass caused intestinal obstruction. Both cases recovered after adequate drainage. The case reported by Warner and Swan is a most unusual one and would seem to demonstrate that fistula formation is not essential for gallstone ileus. In this patient a cholecystectomy had been performed $2\frac{1}{2}$ years prior to the onset of the intestinal obstruction. Three years prior to the cholecystectomy the patient had a sub total gastrectomy performed. Thus $2\frac{1}{2}$ years after the cholecystectomy and $5\frac{1}{2}$ years after the gastric resection, the patient developed acute intestinal obstruction which was shown to be due to a large gallstone. The gallstone produced obstruction $2\frac{1}{2}$ feet from the ileocecal valve. The authors suggested that since no fistula nor any possibility of a fistula between the gall bladder and the bowel was present (the gall bladder having been removed), and since the common bile duct was not dilated in any way suggestive of the passage of the stone through it, the only possible site for the formation of the stone was the duodenum or proximal jejunal loop. They postulated that a small stone had entered the duodenum from the common bile duct and had remained in that location for an unknown period of time. There it gradually increased in size. The existing sub total gastrectomy created conditions in the proximal loop which were similar to those in the common bile duct.

Large gallstones may so weight the bowel as to cause volvulus. This is an unusual mechanism of obstruction. Among the unusual types of bowel obstruction caused by gallstones is the case reported by Barney and Gale. The patient had suffered from diverticulosis for many years. She inadvertently ingested a steak bone which was in the process of being excreted through the flexures of the colon when her solitary large gallstone passed from its cholecystoduodenal fistula and lodged in and obstructed the upper portion of the jejunum. With the onset of the intestinal obstruction and the vomiting and associated intraluminal pressure increase the steak bone perforated the pre-existing sigmoid diverticulum. An abscess was produced which was responsible for paralytic ileus.

Arnold reported a case in which the upper jejunum was completely obstructed by a large gallstone and had in addition herniated into the paraduodenal fossa further increasing the obstruction. This is another example of two etiologic factors operating simultaneously. Either of these factors acting independently could obstruct the bowel.

Gallstones are frequently multiple. For this reason a thorough search of the gastro-intestinal tract should be made in any case of intestinal obstruction caused by a gallstone. It is not rare to have a recurrence of intestinal obstruction after the removal of a gallstone because a second stone has been left. There are many reports in the literature describing this oversight. Noskin and Tannenbaum removed a second stone causing an obstruction 13 months after the first obstruction due to a gallstone. Whenever a large facet facing upwards is found on a gallstone a second stone must be presumed to be present until proved other



FIG. 82. Intestinal obstruction due to gallstones. This is a good example of multiple gallstones. Note gallstone in the terminal ileum and residual stones in the remnant of the gall bladder.

wise. The collapse of the bowel distal to the gall stone obstruction does not preclude the possibility of a second stone being found distally.

In addition to gallstone ileus in which the stone *per se* is the obstructing mechanism, biliary tract disease may cause intestinal obstruction due to other mechanisms. Perforation of the gall bladder results in a peritonitis with a paralytic ileus. In lowly perforating gall bladders, a walled off pericholecystic abscess may result. This can produce intestinal obstruction by virtue of the paralytic ileus commonly resulting from intra abdominal inflammatory processes or a mechanical obstruction may occur due to an adhesion between a loop of bowel and the abscess. When abscesses of large size develop, intestinal obstruction may occur due to the mechanical pressure of the abscess. Blain and Harkins reported an incidence of 0.3 per cent for perforations in a series of 11,794 gall bladder operations. In a similar study they reported an incidence of 0.01 per cent for perforations of the gall bladder in a series of 19,274 autopsies. A study by Cowley and Harkins revealed an incidence of 2.8 per cent for perforations in 12,913 gall bladder operations. The highest incidence for perforations was reported by Mentzer at 12.1 per cent. Twenty three per cent of all gall bladder perforations caused intestinal obstruction. In addition to the obstruction that occurs with or shortly after gall bladder perforation, intestinal obstruction may occur as a late sequel due to an adhesion that resulted from the inflammatory process.

Gallstone ileus may manifest itself in three different forms. In the acute form, the characteristics of acute mechanical intestinal obstruction make their appearance. A second form is the intermittent attack of biliary colic and intermittent intestinal obstruction which releases itself spontaneously. Then finally an acute attack of intestinal obstruction supervenes upon this intermittent process. The third form is the late variety in which the patient is asymptomatic after a brief episode of upper abdominal distress and then the stone is passed per rectum. A history of cholelithiasis or cholecystitis is not always obtained.

The signs and symptoms associated with gall stone ileus depend upon the point of obstruction in the bowel. Those obstructions of the small bowel

which are up as high as the duodenum or jejunum would cause signs and symptoms of high intestinal obstruction. Vomiting and dehydration with electrolyte imbalance play a prominent part in this process. Obstructions which occur in the terminal ileum are by far the most common and present the findings of low intestinal obstruction. These patients do not appear to be acutely ill immediately. Distention appears late in the course of the obstruction as does the vomiting. Despite the common cholecystoduodenal fistula or the cholecystoenteric fistula, jaundice is rarely found in these patients. Few cases are diagnosed pre operatively.

A diagnosis of gallstone ileus may be difficult. In reviewing the literature it is apparent that a correct pre operative diagnosis is seldom made. However, Rigler, Borman and Noble reported in 1941 that in a series of 14 cases of gallstone ileus a correct diagnosis could be made radiologically in 13 of the cases. They emphasized the specific signs which permit a radiologic diagnosis of this type of obstruction.

1. The presence of gas or contrast medium in the biliary tract.

2. The radiologic evidences of intestinal obstruction.

3. Direct or indirect visualization of the calculus.

In addition to these signs, the most important factor is a constant awareness of gallstone ileus as a possible cause for intestinal obstruction in the 60 to 70 year old group and a careful search for evidence corroborating this suspicion. Evidence to be looked for includes (1) a stone in the gall bladder which was formerly noted and has disappeared with the onset of the signs of bowel obstruction and (2) a history of one attack of gallstone ileus. The length of time elapsing between the passage of the stone into the bowel and the development of the obstruction may permit the fistulous tract to heal. As a result only adhesions remain between the gall bladder and the bowel. Angle reported one case in which the gallstone had been present in the bowel for one year and in two other cases the stone had been present in the bowel four months before obstruction set in. Treves reported a case in which the gallstone remained in the bowel for 10 years. A long latent period be



FIG 83 Although the radiopaque gallstones did not visualize within the abdomen (see Figure 92) when the intestines are removed and X rayed in this fashion the biliary calculi are readily apparent. This is a good example of the difficulty in diagnosing small bowel obstruction due to gallstones when one depends solely upon the finding of a radiopaque calculus on an abdominal survey film.



FIG 84 Obstruction at the ileocecal valve due to an impacted gallstone (see arrow)

tween the fistula and the onset of the obstruction may be sufficient to allow the fistula to obliterate itself and therefore air or contrast media would not be expected to be found in the biliary radicles in such cases.

Once the diagnosis has been made immediate surgical treatment is indicated. Harvey in 1888 described a treatment for gallstone ileus in which he opened the abdomen and gently milked the stone down through the ileocecal valve and up into the colon. Following this the abdomen was closed. The stone was passed five days later. Kappel used the same method of milking the obstructing stone through the ileocecal valve in two patients. He believed that if the surgeon were gentle in his manipulations so that the wall of the bowel was not injured and if the loop of bowel in which the stone lay showed no evidence of pressure damage an attempt should be made to milk all the stones through the ileocecal valve. At times stones which successfully pass into the colon become impacted and obstruct the sigmoid colon. This only occurs

in those cases in which the pelvic colon is narrowed due to adhesive bands, pressure from pelvic organs or stenosing lesions of the colon. The procedure most commonly used in the removal of such stones consists of longitudinal enterotomy, removal of the stone and closure of the enterotomy transversely so as not to narrow the lumen. Most surgeons are agreed that the stone should preferably be milked proximally into a relatively healthy portion of the bowel before enterotomy is performed for its removal. It is generally considered unwise to deal with the cholecystoenteric fistula at the time of surgery for the intestinal obstruction. A careful search should be made for other stones in the bowel. In most cases the surgical treatment for removal of a gall bladder and the elimination of the fistula is unnecessary. In all cases follow up gall bladder X ray studies should be made to demonstrate the possible presence of other stones. If found cholecystectomy is indicated at a later date.

The mortality rate for intestinal obstruction due

a gall tone skin ranges from 33 to 93 per cent with a general average of 50 per cent. In contrast to this McLaughlin and James in a serial series of 130 consecutive cases of acute intestinal obstruction from gall tones reported no mortality. The age of the patient, delay in making diagnosis and poor condition at the time of surgery are among the important factors responsible for the high mortality rate.

Enterolithiasis

This is one of the more unusual causes of intestinal obstruction. Williams reported the first case of fecal obstruction of the small bowel in 1908. Scully and Stell reported a case of enterolith small bowel obstruction in 1937. A chemical analysis of the enterolith revealed it to be 72 per cent casein. The rarity of this type of intestinal obstruction is due to the fact that the small bowel physiology normally does not cause marked water absorption. (Some water is absorbed in the small bowel but only to a minor degree. Hence the contents of the small bowel are liquid.) In the colon where water is absorbed fecal impactions and enterolithiasis would not be uncommon. In the case reported by DeWitt, Morrissey and Fuller the enterolith was found to be insoluble in alcohol, an unusual finding, since most enteroliths are soluble in alcohol. The gross pathologic characteristics revealed the enterolith to be made up of inspissated fecal material with some intermingled vegetable fibers. The matting of the vegetable fibers accounted for the formation of the primary calculus.

Retroperitoneal Tumors

The connective tissue tumors which are found in the retroperitoneal area of the body are rather uncommon. The most frequent are the retroperitoneal lipoma, retroperitoneal sarcoma, and retroperitoneal ganglioneuroma.

The retroperitoneal lipoma is a mixed tumor consisting predominantly of lipomatous tissue plus myxomatous or other types of connective tissue. These tumors may attain tremendous size without producing any symptoms. Intestinal obstruction from this type of tumor is rare. Partial intestinal

obstruction characterized by gas pains, and bloating is common.

The retroperitoneal sarcoma which is really a fibrosarcoma behaves in a similar fashion. These growths may produce intestinal distention or impairment of intestinal motility by involvement of the sympathetics. The result may be a reflex ileus such as occurs with retroperitoneal hemorrhage. These tumors are characterized by the fact that they can attain great size with little or no associated symptoms. Colonic compression may occur with resulting obstruction.

Retroperitoneal ganglioneuroma was first described by Loretz in 1870. Although his case was of the mediastinal variety, it called attention to the pathologic entity. It was not until 1898 that Busse described a pelvic ganglioneuroma. Numerous case reports describing this type of tumor have appeared since. Up to 1946 over 200 cases of this type were reported. These tumors are believed to be congenital in origin. The nidus for their development is cells displaced during the migration of ganglionic crests in embryonic life. In man the sympathetic ganglion organization is the result of the migration of individual cells from the neural crest substance down the dorsal nerve roots and peripheral trunks to form paired ganglionic clusters.

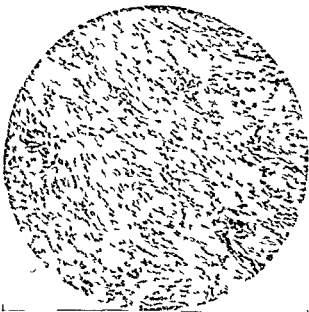


Fig. 83 Retroperitoneal ganglioneuroma. Magnification $\times 125$.

GASTROINTESTINAL OBSTRUCTION



Fig. 66 Retroperitoneal ganglioneuroma. Magnification $\times 60$

ters dorso-lateral to the aorta. These cells may never reach the ganglionic mass and may become arrested or displaced anywhere along the way. Such cell rests may remain quiescent forever and never give rise to trouble or they may begin to proliferate at any period of prenatal or postnatal life. The type of tumor that is formed by these cells depends upon the level of differentiation that has been attained before neoplastic development took place. Although acute small bowel obstruction does not occur as a result of these tumors, partial intestinal obstruction is quite common. Characteristically these patients present a large abdominal tumor mass with a history of gas pains or bloating. Many give a history of nausea but no vomiting. Constipation is apt to be prominent. This may be the result of colonic compression or the effect of sympathetic stimulation.

Mesenteric Tumors and Cysts

Mesenteric cysts and tumors are uncommon causes of acute small bowel obstruction. In the majority of reported cases, the cystic tumors were asymptomatic. The visible presence of a tumor mass is often the first indication of its presence. The most common complication is intestinal obstruction. In such cases, surgical intervention is

undertaken for the complicating process and the true diagnosis is not made until the abdomen is opened. At times, such cysts may reach large proportions. They may become incarcerated in the pelvis and produce obstruction there due to pressure. Some form of intestinal obstruction has been reported in 40 to 50 per cent of all cases of mesenteric cysts. The treatment is surgical in all cases. The signs and symptoms depend upon the level at which the bowel is obstructed and are no different from those of any other type of mechanical obstruction except that a readily palpable, freely movable abdominal mass is present. A history of the presence of an asymptomatic mass of this type prior to the onset of bowel obstruction should lead one to suspect that a mesenteric cyst is involved.

All the cystic tumors that develop between the leaves of the mesentery are classified as mesenteric cysts. This descriptive name is applied to such growths regardless of their histologic type. Since all these growths produce the same symptoms and present much the same physical findings and require the same treatment. The tumors develop in the space between the leaves of the mesentery, the mesocolon, mesosigmoid and the omentum. The area is called the mesenteric space. Normally,

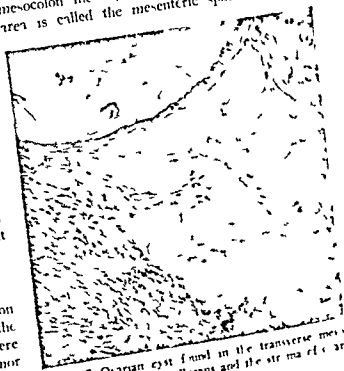


Fig. 67 Ovarian cyst found in the transverse mesocolon. Note the corpus allicans and the struma of the follicles.

this space contains fat, arteries, veins, lymph vessels, and lymph gland. Aberrant ovarian, splenic, or thyroid tissue may be found between the layers of the mesentery, and thus may be classified as mesenteric growths. The Wolffian body and duct, the Mullerian body, and the germinal epithelium may have retroperitoneal sequestrations. As the layers of the mesentery are separated by growths between them, the space increases in size. Solid tumors such as fibromas, lymphomas, metastatic carcinomas, and hemangiomas may come to occupy this mesenteric space; however, none of these are classified as mesenteric cysts.

Bennett, an anatomist, recorded the first description of a cystic tumor of the mesenteric space in 1707. Prior to the 19th century, all such cases were reported by anatomists. In the late 19th and 20th centuries, as a result of the widespread practice of abdominal surgery, a new era began in which these growths were described by surgeons. Although in the early years a correct diagnosis was not made prior to the opening of the abdomen, in recent years an increasing number of correct diagnoses have been made prior to surgery.

Although the rarity of such cystic growths is indicated by the fact that only 2 such cases were noted out of 129,748 admissions to Grace Hospital in a 6-year period, nonetheless 1000 such cases have been reported in the world literature.

The developmental processes by which these cystic tumors develop are controversial. No one concept can explain all the types of mesenteric cysts. A classification which rather fully describes and explains the mesenteric cyst has been presented by Loeb as follows:

A. Embryonic cysts (a) enterostomas of intestinal origin

- 1 Due to sequestration during the embryonic stage from omphalomesenteric duct or embryonic diverticulum
- 2 From persistent Meckel's diverticulum
 - Either (1) or (2) by the extravasation of lymph blood or by neoplastic change may become
 - (a) Chylous cyst
 - (b) Hematogenous cyst
 - (c) A malignant cyst
- (b) Embryomas spring from retroperitoneal organs within the mesenteric space. These are
 - (a) Wolffian duct and body
 - (b) Mullerian body
 - (c) Germinal epithelium

- B. Teratomatous cysts. These arise from misplaced ovarian tissue in the mesenteric space.
- C. Infectious cysts. These arise as a result of infection by
 - (a) Echinococcus
 - (b) Tuberculosis
 - (c) Encapsulated abscess

In reviewing 200 cases of mesenteric cysts, Burnett, Rosemond, and Pucher reported the following sites of origin for these cystic structures:

1 Mesentery of small bowel	46.5%
2 Mesentery of sigmoid colon	15%
3 Mesocolon	11%
4 Mesentery of cecum	8%
5 Mesentery of descending colon	2.5%
6 Mesentery of appendix	1.5%
7 Omentum	2%
8 Gastrophilic omentum	0.5%
9 Duodenum	0.5%
10 Retroperitoneal	5%
11 Unknown	7.5%

In this series a mortality rate of 15 per cent was reported if resection of the bowel was required. This is approximately the figure reported throughout the country for intestinal obstruction. The surgical management of small bowel obstruction due to mesenteric cysts differs from that when the obstruction is due to other causes. It depends upon whether surgery was undertaken for the primary disease or for the complicating bowel obstruction. If surgery is undertaken for the primary disease, excision of the cyst is considered to be the treatment of choice. In many cases the cyst has so encroached upon the arterial as well as the venous blood supply to the overlying segment of bowel that strangulation of the bowel has occurred. In such instances, resection of the bowel and the mesentery containing the cyst must be performed. Often the blood supply to the overlying segment of bowel is compromised during the process of excision of mesenteric cysts uncomplicated by obstruction. In this event, resection of the bowel thus deprived of its blood supply is the only safe procedure possible. An end-to-end anastomosis is preferable in all such cases. In the poor risk patient, marsupialization and exteriorization of the bowel may be required as an emergency measure. The method of perforation and aspiration as well

as puncture of such cysts with aspiration of their contents has no place in the modern treatment of this disease

In most instances the mesenteric cyst will be found to have produced a considerable enlargement of the mesenteric space. The enlarging cyst pushes the blood vessels toward one leaf of the mesentery. As a result one side of the mesentery is almost avascular whereas the other leaf contains all the mesenteric vessels. This permits the surgeon to perform an almost bloodless operation by incising the peritoneal cover over the cyst on the avascular side and then shelling out the tumor from the mesenteric space. In an occasional case the cyst may be found to be dumbbell shaped presenting a large mass bulging out on both sides of the mesentery. In cases of this type resection of the overlying bowel with end to end anastomosis may be required. It must be remembered that the cyst should be removed in addition to correcting the bowel obstruction.

In this group all patients who are subjected to surgery should be well prepared with blood so that optimum conditions prevail at the time of operation. At least 1000 cc of blood should be available in the operating room because of the well known tendency of mesenteric and retroperitoneal tumors to bleed. This is especially true of the retroperitoneal tumors whose removal is so often complicated by shock due to autonomic trauma.

Infections

There are varied types of infection within the peritoneal cavity capable of causing either acute mechanical intestinal obstruction, sub-acute or chronic intestinal obstruction or paralytic ileus. The mechanism of obstruction produced by the infectious process varies from those caused by adhesions by compression of an abscess by formation of a granuloma by edema of adherent bowel or by paralytic ileus. The obstructions associated with intra abdominal infections are usually incomplete and are rarely of the strangulating variety. As a result this group of bowel obstructions should be treated by intestinal intubation using the long intestinal decompression tube. The liberal use of antibiotics and our knowledge of electrolyte balance have their most effective role



Fig. 88 Paralytic ileus secondary to tubo-ovarian abscess

in the management of obstructions in this group. It must be constantly borne in mind however that any of these obstructions may become complete necessitating surgical intervention. Aside from such isolated cases we believe that this group of inflammatory abdominal obstructions should always be given a trial period under conservative management. In this fashion most of the patients in this group can be carried over the acute process without surgery. An appreciable percentage of the patients may require surgery at a later date for treatment of the sequelae of such inflammatory processes. Of the sequelae band adhesions producing bowel obstruction are the most common.

Appendicitis repeated infections of the appendix not infrequently produce intestinal obstruction by virtue of the fact that such an appendix may become fixed transversely across the terminal ileum by fibrous adhesions following the subsidence of the acute inflammatory process. The compression of the ileum may obstruct it. On the other hand the appendix may become adherent to

a loop of ileum several feet from the ileocecal valve forming a bridge. In such instances a second loop of bowel may herniate under the bridge so formed causing acute intestinal obstruction. Acute appendicitis *per se* is uncommon as a cause of intestinal obstruction. Buckwalter and Modlin reported a case of this type in which the acutely inflamed appendix had become adherent by its tip to the posterior parietal peritoneum. The acutely inflamed appendix had crossed over the terminal ileum completely obstructing it. The base of the appendix had perforated.

Peritonitis. Intestinal obstruction in association with peritonitis may be varied in its mechanism. The most common variety of intestinal obstruction associated with peritonitis is paralytic ileus. This may be confused with the paralytic ileus that can follow operative trauma. This latter ileus occurs after surgery and is due to a reflex splanchnic stimulation which inhibits intestinal motility. Ileus of this kind may be due to anesthesia, manipulation of the bowel or any procedure in which the bowel is opened or sectioned. It can usually be

cleared up by the use of a long intestinal decompression tube. The tube need not proceed down the gastro intestinal tract for treatment to be effective. The suction applied to an indwelling gastro duodenal tube is often sufficient because it removes the swallowed air which is responsible for most of the intestinal distention in this type of case.

The paralytic ileus associated with peritonitis is a more serious problem. The silent abdomen found with this type of ileus generally occurs after the third postoperative day at a time when the peristaltic activity should normally have returned. The silent abdomen is associated with generalized or localized peritonitis. Sometimes a single loop of bowel or the entire gastro intestinal tract may be inhibited. By the fifth day there may be a matting together of loops of bowel by the plastic exudate of the peritonitis. A mechanical obstruction may be superimposed upon a purely paralytic ileus as a result.

An additional variety of mechanical obstruction may occur due to kinking of a loop of bowel to or around an inflammatory mass or an abscess. Occa-



FIG 89 Paralytic ileus following abdominal surgery. Note small and large bowel distention.



FIG 90 Section of ileum showing ileitis with ulcer formation and perforation of the ulcer. Magnification $\times 125$.

sionally such intra abdominal abscesses may reach a size sufficiently large to obstruct the small bowel by compression. The adherence of a loop of bowel to a pelvic abscess may cause an inflammatory reaction in the small bowel wall which will obstruct it.

The obstructive symptoms in all these varieties subside when the abscess is drained or when the inflammatory process subsides. Since strangulating obstructions are uncommon with this type of obstructive process nonsurgical treatment using the long intestinal decompression tube liberal use of antibiotics and adequate and proper intravenous alimentation may be utilized. This overcomes the inflammatory process permitting the obstruction to disappear.

The hepatic flexure of the colon and the small bowel are common sites for intestinal obstruction in the presence of perforation of the gall bladder. This may or may not be associated with pericholecystic abscess. In addition to the early cases of intestinal obstruction following perforation with a



FIG. 92 The same patient as in Figure 91. Note the right leaf of the diaphragm pushed well up into the chest.

walled off abscess obstruction of the small bowel may follow as a late sequel due to the adhesions that have been left.

Poliomyelitis. Poliomyelitis may produce a severe type of paralytic ileus which simulates acute intestinal obstruction. In some cases the stomach becomes so dilated that on a survey film of the abdomen it may easily be confused with a cecal volvulus. Three to four thousand cubic centimeters of fluid may fill such dilated stomach. A diagnosis of paralytic ileus should be suspected in any patient known to have poliomyelitis who suddenly becomes distended and begins to vomit. The passage of a long intestinal decompression tube adequately weighted with mercury and the institution of continuous suction is the only treatment. A survey film taken before and after such gastric decompression clarifies the diagnostic problem by proving that the greatly dilated fluid-filled portion of the gastrointestinal tract is 1 inch.

Tuberculosis. Intestinal tuberculosis is clinically recognizable in two forms: (1) the ulcerative and (2) the hypertrophic. The ulcerative type



FIG. 91 Huge subphrenic abscess with intestinal obstruction as a result of pressure. Note the large radiopaque mass in the right upper quadrant.

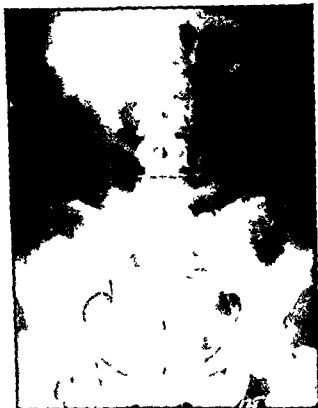


FIG 93 Patient C 11 age 43 female Small bowel obstruction produced by an acute ulcer of the ileum This is the same patient from whom the section in the microphotograph in Figure 90 was taken



FIG 94 Sarcoidosis producing small bowel obstruction Magnification $\times 150$

is usually secondary to pulmonary tuberculosis although in children it may be primary as a result of drinking tuberculous milk. The ulcerations are most common in the ileocecal area but may also be found proximal or distal to this point. This disease is recognizable by the ulcers produced in the mucosa. The overlying peritoneum is thickened so that perforation is uncommon. The extension of the inflammatory process to the serosa results in adhesions. These may contract and produce acute kinks of the bowel, a common cause for obstruction in such cases. When ulceration and destruction are considerable the scar formation is proportionately great. In the encircling type of ulceration the cicatrization may result in a stenosis of the bowel. Such stenosis is likely to lead to chronic or partial intestinal obstruction rather than to acute bowel obstruction which occurs as a result of kinks in the bowel.

The hypertrophic type of tuberculosis is gener-

ally found in the terminal ileum and the ileocecal region and is a disease of young adults. Clinically this disease is gradual in onset and simulates intestinal carcinoma. Pathologically there is a considerable formation of granulation tissue in the submucosa and subserosa. The mucosa projects into the lumen of the bowel greatly narrowing it. The involved portion of bowel is stiff and thick and presents a tumor like mass difficult to distinguish from carcinoma. As an added confusing factor the regional lymph nodes may be enlarged.

Chronic Granuloma In rare instances chronic granulomatous masses of undetermined origin may be found in the region of the terminal ileum obstructing it. The symptoms which are those of high grade partial intestinal obstruction are of progressively increasing severity. The marked narrowing of the terminal ileum found with terminal ileitis may be classified in this group. The lumen of the bowel may be narrowed to such a degree that a stringlike lumen is readily demonstrable in the upper gastro intestinal series. The proximal bowel is markedly dilated and its wall hypertrophied in a compensatory mechanism to overcome the obstruction.



FIG. 95 Foreign body granuloma producing intestinal obstruction. Note the giant foreign body cells.



FIG. 96 Segmental ileitis producing small bowel obstruction. Notice the marked narrowing of the small bowel; this is the strong sign.

Retroperitoneal Hemorrhage

Spontaneous retroperitoneal hemorrhage is a rarely encountered lesion. Up to 1945 only 26 such cases were reported. Since then, however, a number of cases have been reported. It is seldom diagnosed pre-operatively because the possibility of retroperitoneal hemorrhage is not considered in the differential diagnosis. As has been pointed out, the diagnosis can be made pre-operatively with a reasonable degree of certainty if one considers such hemorrhage as a possible cause in any case of ileus of unexplained origin. The retroperitoneal bleeding gives rise to dull sickening pain of sudden onset which may be persistent or subside gradually. It is usually associated with nausea and vomiting. The patients are restless and apprehensive. If the hematoma ruptures into the peritoneal cavity, there is sudden severe pain and shock. In the cases reported to date, 76 per cent of all the adults with spontaneous retroperitoneal hemorrhage presented generalized arteriosclerosis or hypertension or both.

Peritoneal aspiration as a method of diagnosis is being advocated to distinguish between strangulating and nonstrangulating obstructions; the presence of a bloody fluid being the finding of importance. The presence of such bloody fluid in the peritoneal cavity when associated with the signs of intestinal obstruction is said to be pathognomonic of a strangulating obstruction, whereas the finding of a serous fluid is said to indicate a nonstrangulating obstruction. This point is emphasized at this time because retroperitoneal bleeding may also present all the findings of intestinal obstruction and in addition a bloody fluid within the peritoneal cavity. A similar finding may occur with torsion of a pedunculated ovarian cyst and strangulation, ectopic pregnancy, hemorrhagic pancreatitis, and ruptured follicle cyst of the ovary. The one point to be remembered in the differential diagnosis between all these lesions and a strangulating intestinal obstruction is the fact that only in the case of strangulating obstruction is the blood-tinged or bloody peritoneal fluid associated with the signs of a mechanical intestinal obstruction. All the other varieties of bloody fluid described are associated with a paralytic ileus. The hyperperistaltic, colicky pain associated with for

horygmus and the history of onset should be sufficient to differentiate the lesions present. Neglected strangulating obstruction seen by the surgeon so late in the course of the disease that an almost silent distended abdomen is found may create a diagnostic problem.

Hemorrhage into the retroperitoneal area may cause intestinal obstruction in two ways. First a paralytic ileus may occur as a result of the presence of blood in this area and secondly the hemorrhage may be so massive that compression of the second portion of the duodenum may result. Cases of this type were reported by Sachs and Macht and by James. In the case report of James the blood clot was successfully evacuated releasing the duodenal obstruction. In the case of Sachs and Macht a gastroyejunostomy procedure was necessary because of the impossibility of relieving the mechanical compression of the duodenum by evacuating the blood clot.

In any case of retroperitoneal hemorrhage once the danger of immediate death by exsanguination has passed the complication of paralytic ileus which is a serious problem must be treated by intestinal intubation. The exact mechanism by which this ileus is produced is not completely understood. Cole believes that these patients develop symptoms from a central nervous system reflex caused by irritation of the sympathetic system. Some authors are of the opinion that the ileus is the result of a paralysis of both the sympathetic and parasympathetic systems.

Retroperitoneal hemorrhage may be classified into three large groups: (1) traumatic, (2) operative, (3) nonoperative and nontraumatic. In the traumatic group may be found all those cases of injury to the kidneys, muscles, blood vessels and other retroperitoneal structures. In addition fractures of the ribs, vertebrae and pelvis may produce such bleeding.

The operative causes are secondary to any operation performed in the retroperitoneal area.

The nonoperative and nontraumatic group constitutes by far the largest group of cases. Here we find all cases of retroperitoneal hemorrhage due to diseases of the kidneys such as cysts, calculi, tumors and infections; diseases of the blood such as leukemia, hemophilia and thrombocyto-

penia; diseases of retroperitoneal blood vessels such as aneurysms, spontaneous rupture of vessels due to arteriosclerosis and hypertension or diseases of the retroperitoneal tissues such as tumors, infections and emboli. Such retroperitoneal hemorrhage may occur during the last trimester of pregnancy following intercourse or bowel movement. In these cases the bleeding may begin in the broad ligament and dissect its way upward and medially. Diseases of the liver such as cirrhosis may occasionally be associated with bleeding retroperitoneally.

In addition to all of these there is a small group of cases in which no actual pathologic causative mechanism is demonstrable.

Small Bowel Tumors

Small bowel tumors are uncommon causes of intestinal obstruction. There are four possible mechanisms by means of which the tumor mass may produce obstruction:

1. It may obstruct the bowel lumen by virtue of its size.
2. The tumor may act as a nidus causing intussusception.
3. Tumors of large size and subserosal position may cause volvulus of a segment of bowel.
4. Malignant tumors may cause adherence of adjacent loops of bowel, sharply angulating and obstructing them.

The small bowel tumors fall into three large groups: (1) benign tumors, (2) malignant tumors, and (3) tumors pathologically benign but metastasizing such as carcinoids.

The symptoms of small bowel tumors depend upon the size, location and behavior of the tumors. All varieties of intestinal obstruction may be encountered ranging from acute, subacute, chronic or acute exacerbation of a chronic obstruction. In any type the obstruction may be partial or complete, intermittent or progressive. The sudden onset of obstruction may be preceded by a long interval of abdominal complaints of a vague or mild nature. Generally the diagnosis of small bowel tumor is made with difficulty. The symptoms of benign and malignant tumors do not differ greatly since in either case they are dependent upon the ability of the tumor to produce bowel

obstruction or an interference with the continuity of the gastrointestinal tract. Because of the length of the small bowel and the overlapping of loops the radiologic study is not nearly as helpful diagnostically as it is elsewhere in the gastrointestinal tract. For this reason negative results in radiologic studies do not rule out the presence of a small bowel tumor. Positive results are of great value—this is particularly true of the observation of a point of narrowing associated with a proximally dilated bowel.

Although the greatest number of cases of small bowel tumors are found in the fourth and fifth decades of life, no age group is exempt. Tumors have been described in children as young as 4 years and in patients 83 years of age.

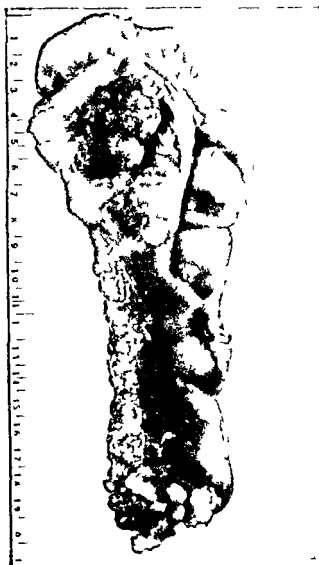


FIG. 97. Cross specimen of small bowel tumor producing obstruction.

The obstruction produced by small bowel tumors requires surgical intervention. Primary resection and anastomosis is the procedure of choice. Conservative management has no place in the treatment of this type of obstruction.

Benign Tumors. All types of benign tumors have been reported to produce intestinal obstruction. Among those reported are adenoma, polyp, myoma, fibroma, lipoma, leiomyoma, hemangioma, lymphangioma, neurogenic tumors, and pancreatic rests. The first authenticated description of a leiomyoma of the small bowel was the report of Loerster in 1855. Up to 1941 only 210 leiomyomas of the jejunum had been reported. All age groups were represented in the cases reported. The leiomyomas were reported in all portions in the bowel wall: (1) intraluminal, (2) intramural, (3) extraluminal. The intraluminal type of tumor is pedunculated or polypoid. It may produce intestinal obstruction either by its mass filling the lumen or by causing an intussusception. In the first type the symptoms would be those of a partial intestinal obstruction of gradual onset. In the latter type the symptoms would be of long duration and then



FIG. 98. Small bowel tumor with obstruction. This is a radiograph taken prior to surgery.



FIG 99 Leiomyofibroma of the ileum producing in partial intestinal obstruction. This patient was a 15 year old white male



FIG 100 The same specimen as shown in Figure 99. Note the tremendous hypertrophy of the proximal bowel. There is also a tremendous dilatation of the bowel proximal to the point of obstruction. This is indicative of a low grade obstruction of long duration which finally became acute.

a sudden acute complete intestinal obstruction would occur. This complication is particularly dangerous because strangulation of the intussuscepted bowel may appear with little or no clinical evidence of strangulation. The abdomen may be found to be relaxed and not tender. This is due to the fact that such infarcted bowel (intussusceptum) is ensheathed within a normal liver of bowel.

The intramural type of leiomyoma being of slow growth produces signs and symptoms of partial intestinal obstruction of variable degree. This progresses very slowly with the continued growth of the tumor.

The extraluminal leiomyoma protrudes into the peritoneal cavity. It does not encroach upon the lumen of the bowel. It may attain great size before it produces symptoms. A large tumor mass may be palpable without causing any discomfort to the patient. In this position the downward drag of the tumor may produce bowel obstruction by kinking or angulating the bowel or the tumor may compress the bowel against the abdominal wall obstructing it. In isolated instances volvulus of the

loop of bowel with this tumor has been reported.

Since they are histologically benign leiomyomas are classified as benign growths. However 16 per cent have been reported to become malignant. Among the leiomyomas thought to be histologically malignant 15 to 30 per cent are found to metastasize. The liver is the site of predilection for such metastasis.

These tumors are radioresistant. For this reason treatment must be limited to surgical removal. Since they are of low grade malignancy a wide primary resection and end to end anastomosis generally presents a good prognosis. Occasional cases may have so infiltrated the circumjacent tissues that adequate removal is impossible.

Jejunal polyps commonly produce intestinal obstruction because of intussusception. Jejunal polyps and intussusception associated with abnormal melanin pigmentation is uncommon. Peutz in 1921 first described a familial syndrome consisting of intestinal polyposis associated with pig

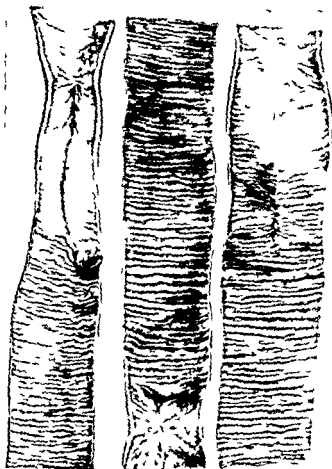


FIG. 101. Lipoma in the small bowel causing intussusception. Note the points of stricture formation as a result of the intussusception. Because of its length the specimen has been drawn in three sections: the top of the second strip should be joined to the bottom of the first and the top of the third to the bottom of the second to reconstruct its continuity.

mentary changes of the buccal mucosa, lip, and skin. Jeghers introduced this syndrome to the American literature in 1944. The important features of this syndrome are: distinctive melanin spots of the buccal mucosa and lips, face, and digits. The mouth pigmentation is the *sine qua non* of this portion of the syndrome and is associated with polyposis of the small bowel. The stomach, colon, and rectum may be involved, but the presence of polyps in the small intestine is the constant feature of this syndrome. The polyps, either single or multiple, are usually found in the jejunum. Each of the patients required surgery on one or more occasions for episodes of intussusception. The syndrome seems to be inherited as a simple Mendelian dominant. Of the 610 cases of intus-

susception reported by Gross and Ware, less than 1 per cent could be attributed to polyps.

Other types of benign small bowel tumors occur with decreasing frequency down to the lymphangioma and melanoma that are the least common. The diagnosis is seldom made preoperatively. In these cases, intestinal obstruction is usually incomplete with the possible exception of those patients who develop intussusception. Melanotic tumors are rare small bowel lesions. McCollum found only 33 cases of this type reported. The ileum appeared to be the most common site of involvement with the jejunum second and the duodenum last. Intussusception producing bowel obstruction is a common complication.

Lymphangioma is the least common small bowel tumor. In a review of the literature in 1923 Gravelle and Green found only 13 cases reported. All these were in the nature of simple case reports. These tumors are thought to be due to dilatations of the lymphatics in the submucosa. Some of them may be the result of congenital dilatation or strictures in the lymph conducting system. To date no malignant change has been reported in this lesion. Although many of the lymphangiomas are asymptomatic, the most common symptom is pain due to obstruction.



FIG. 102. Lymphangioma producing obstruction at the terminal ileum. Magnification $\times 120$.

Malignant Tumors Carcinoma of the small intestine is the most common malignant tumor in this portion of the gastrointestinal tract. Other malignancies are malignant myomas and sarcoma. Sarcoma is further broken down into myosarcoma, fibrosarcoma, myxosarcoma, and lymphoblastoma. The lymphoblastoma is further subdivided into lymphosarcoma and Hodgkin's disease.

The first description of carcinoma of the jejunum was made by Sorlin in 1824. Leichtenstern in 1876 reported the first autopsy series of malignant lesions of the small intestine. He found that the malignancies made up 0.07 per cent of the malignant lesions of the entire gastro-intestinal tract. Carcinoma of the ileum is second in incidence to carcinoma of the duodenum. Primary carcinoma of the jejunum is uncommon. Slightly more than 100 cases have been reported to date. These were chiefly in the upper jejunum within a short distance of the ligament of Treitz. Mayo and Steinberg report one primary carcinoma of the jejunum as a familial incident.

Intestinal obstruction is slowly progressive in these cases. A radiologic diagnosis may be made on the basis of a narrowing in the bowel lumen or a filling defect. Special small bowel studies using the long intestinal decompression tube for bowel localization may be required. Obstruction due to carcinoma of the small bowel may be the result of an occlusion by the carcinoma or of intussusception produced by the tumor. This is most likely when the lesion is polypoid. Adenocarcinoma is the most common type found. Some of these arise from intestinal polyps; they may grow in a polypoid fashion until the lumen of the bowel is completely obstructed or they may invade the muscular and submucous layers to produce early stenosis. Because of the ulceration of the overlying mucosa, some bleeding is usually associated with all small bowel carcinoma. Multiple primary carcinoma of the small bowel is rare; only six



FIG. 103 Adenocarcinoma involving the small intestine producing obstruction.



FIG. 104 The same patient as shown in Figure 103. Note the small bowel distention and the long tube passed prior to surgery. Primary resection and anastomosis can be performed with ease when the long tube is far down the bowel.



FIG. 10b. Polyps of the colon producing intussusception associated with an adenocarcinoma of the colon. This is a good example of the multiple primary etiologic mechanisms by which intestinal obstruction can be produced.

cases having been reported. In all six the tumors were found to arise synchronously in different foci. Nine additional cases were reported in which one carcinoma involved the small bowel while another involved some other portion of the gastrointestinal tract such as duodenum and stomach, duodenum and sigmoid ileum and rectum, jejunum and stomach, ileum and cecum, ileum and sigmoid ileum and cecum and sigmoid. Although multiple primary carcinomas are uncommon at operation for an obstructive lesion due to carcinoma a search should be made for other tumors as

Obstruction of the small bowel due to car-

cinoma should be treated by wide resection and anastomosis. The prognosis is generally poor regardless of the degree of resection. The average duration of life following surgery in such case is 18 months. A better prognosis is possible if the patient develops acute intestinal obstruction due to intussusception early in the course of a malignancy associated with polyps. In such cases prompt surgical treatment for intussusception removes an early lesion.

Sarcoma is an uncommon tumor in the small intestine. Of this group lymphosarcoma is the most common. Over 300 cases have been reported to date. Four recognizable types have been described: (1) a circumscribed tumor independent of Peyer's patches, (2) a polypoid tumor arising in Peyer's patches, (3) a plaque like tumor presenting masses on the serosa, and (4) infiltrating masses in the mucosal fold. Histopathologically two types of sarcoma are recognizable: (1) the

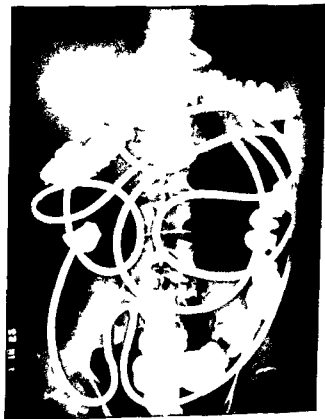


FIG. 10c. Obstruction of the terminal ileum due to a reticular cell sarcoma. In this patient J. S. Ak, 48, wide resection and anastomosis were performed followed by X-ray therapy. The patient expired nine months after surgery.

small round cell type and (2) the reticular cell type with large mature pale staining cells.

The ileum is most commonly involved by sarcoma. Small bowel obstruction is the most common symptom. The circumcribed type of tumor is generally obstructive early whereas the polypoid variety is likely to cause intussusception. The average duration of symptoms before surgery is about eight months and the most common symptom complex is that of slowly progressive intestinal obstruction. In an occasional case perforation of the bowel occurs with resultant peritonitis.

Fibrosarcoma may occasionally involve the small bowel and the colon. In a case noted by the au-

thors multiple fibrosarcomas were found in the small intestine mesentery and the rectosigmoid necessitating the resection of 27 cm of colon and 210 cm of small bowel. The prognosis is generally poor; the average survival time is 22 months regardless of the type of surgery performed. Wide excision followed by X-ray therapy is still indicated for all such new growths.

A sarcoma with a better prognosis is the rare leiomyosarcoma. This is rarely found in the jejunum. In a review of the literature Anderson and Doob found only 10 leiomyosarcomas from 1875 to 1933. Of 108 small bowel malignancies treated at the Mayo Clinic from 1907 to 1939 only 2 were leiomyosarcomas of the jejunum. These tumors are characterized by a slow onset producing vague gastro-intestinal complaints. Bowel obstruction occurs when the neoplasm has involved the lumen or when the growth has reached a very large size.



FIG. 107 Patient E. J. age 39 white female. Multiple fibrosarcoma involving the small intestine mesentery and rectosigmoid. The sarcomatous involvement of the colon in the pelvis was so extensive that excision was thought to be impossible. A colostomy was performed. Note the barium filled colon by injection through the colostomy. The small bowel had become completely obstructed as a result of a tumor growing from the involvement of the colon. A resection of the obstructed small bowel and end to end anastomosis successfully relieved the small bowel obstruction. This was a palliative procedure.



FIG. 108 Same patient as shown in Figure 107. Survey film of the abdomen following surgery to correct small bowel obstruction. Notice the absence of the small bowel distention. The palliative surgery succeeded in making the patient's last days more comfortable.

The degree of malignancy is so low and the rate of invasion so slow that despite its size no evidence of metastasis may be found at the time of surgery.

Lymphosarcoma is the most common tumor arising from the lymphoid structure in the gastrointestinal tract. It may produce a deformity either as a polypoid growth projecting within the lumen of the bowel or as an intramural lesion that infiltrates its wall. Intussusception is not uncommon. Diffuse infiltrating lymphosarcomatous lesions may be found in the distal portion of the ileum. This is their most common site. In contradistinction to carcinoid, there may be multiple sites of involvement with lymphosarcoma and there may be irregular dilatation of the involved segment of bowel rather than constriction. These localized lymphosarcomatous masses in the intestine may attain a very large size. The treatment of choice is extensive excision of the tumor with the removal of the involved lymph nodes. This is then followed by X-ray therapy. The prognosis depends more upon the location of the lesion than upon its histologic type. Recurrence is more frequent with the small bowel lesions than with cecal lesions. Nitrogen mustard has been found to be of some value in the treatment of recurrent lymphosarcoma which has become resistant to further X-ray therapy. Temporary remissions may be obtained. In general, however, the results are disappointing.

Hodgkin's disease is ranked among the rare small bowel tumors causing intestinal obstruction. A huge amount of literature has appeared on Hodgkin's disease since the first report in 1832. Deutsch reported one of the few cases in which Hodgkin's disease caused small bowel obstruction.

Carcinoid Tumors of the Ileum. Carcinoid tumors enjoy the unique distinction of being classed with benign tumors even though they are capable of metastasizing. Metastasis of carcinoid of the appendix is uncommon but it is common in carcinoid of the ileum. The incidence of metastasis from carcinoid of the ileum varies from 18.3 to 52 per cent. In addition to its tendency to metastasize, carcinoid of the ileum commonly produces intestinal obstruction. Small carcinoids of the ileum may cause a symptom complex suggestive of recurrent appendicitis. The obstruction produced



FIG. 109 Carcinoid of the ileum producing intestinal obstruction. Magnification $\times 125$.



FIG. 110 Carcinoid of the ileum producing intestinal obstruction. (Same patient as in Figure 109. In higher magnification.) Magnification $\times 145$.

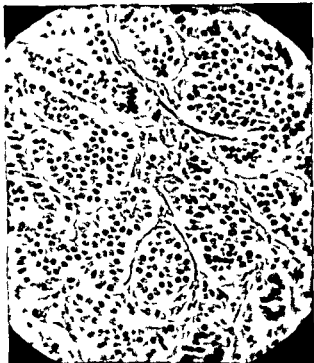


FIG 111 High magnification of carcinoid of the ileum producing intestinal obstruction. Magnification $\times 650$

by carcinoid of the ileum is slowly progressive. There is a long history of abdominal discomfort before the onset of complete obstruction. The signs and symptoms are those found with any low intestinal obstruction. This is characterized by the late onset of distention which becomes a prominent feature of the disease. Vomiting may be minimal or even absent until late. There is no appreciable change in the temperature, pulse, blood count, urine, or blood chemistry. Obstipation sets in after the bowel distal to the point of obstruction has been emptied.

Endometriosis

Endometriosis of the small bowel causing intestinal obstruction is uncommon. Thirty-two cases were reported through 1951. In the majority of cases the terminal ileum was involved. Chronic intestinal obstruction is the commonest manifestation. In 5 per cent of endometriosis cases involving the terminal ileum the invasion is deep and sufficiently extensive to produce a high grade obstruction. It is very apt to be confused with appendicitis and at the time of surgery with car-

cinoma of the ileum. Thickening of the small bowel wall by infiltration and acute kinking due to the dense peritoneal adhesions are the characteristic gross features noted at the time of surgery. It is essential to remember that the adhesions due to endometriosis are stronger than the structures which they unite. Extreme care should be exercised in dissecting these adherent structures in order to prevent perforation.

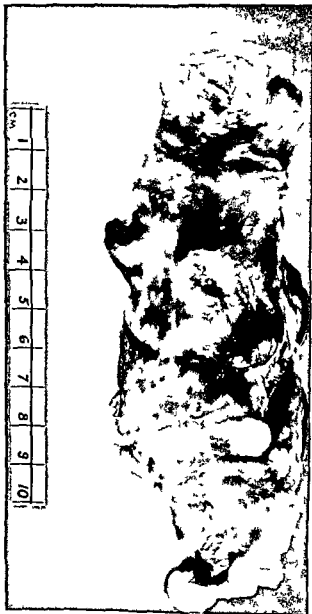


FIG 112 Endometriosis producing obstruction of the terminal ileum



FIG. 113 Endometrio is producing obstruction of the terminal ileum. Note the endometrial glands in the cytotrophoblastic tissue. Magnification $\times 150$.

The most important single feature in the histopathology of endometriosis is the fact that it responds to hormonal stimulation. Unlike the endometrium of the uterus, such endometrial implants lack drainage and a good supporting tissue, as a result the implant burrows beneath the peritoneum along tissue planes and may even invade the lymphatics. Because of lack of drainage such implants accumulate blood thus forming cysts. Secondary implantation occurs by spillage from such ruptured cysts. The adhesions resulting from repeated episodes bind the contiguous structures.

Endometriosis is capable of producing small bowel obstruction in three different fashions:

1. A loop of bowel may become adherent to endometrial masses either in the ovaries or in the cul de sac with a resultant angulation and obstruction.
2. The endometrial implant on the surface of the bowel may produce such extensive bowel involvement that a tumor mass encroaching upon its lumen is formed thus producing obstruction.
3. Endometrial implant on the surface of the bowel may produce fibroblastic proliferative

changes with adhesions of such density that the small bowel becomes adherent to other loops or to the pelvic structures producing angulation and obstruction.

Meyer in 1909 reported the first detailed description of surgery for the relief of an obstructive lesion due to endometriosis. Mount reported the first case in which stricture of the ileum was caused by misplaced endometrial tissue in 1920. This was followed by isolated case reports.

The treatment of endometriosis with obstruction must be highly individualized. It depends upon the degree of bowel involvement, the age of the patient and her desire or ability to bear children and the presence or absence of pelvic involvement. Unlike endometriosis of the colon, small bowel endometriosis generally occurs in young women. Most of these have never been pregnant or have had only one pregnancy. In such cases, if there is little or no pelvic involvement, resection of the ileum and end-to-end anastomosis is sufficient. Such patients are urged to become pregnant at the earliest possible time. If there is extensive endometriosis of the pelvic viscera in older women, the procedure of choice is total hysterectomy and bilateral oophorectomy and hysterectomy may be constructed by a cauterizing lesion if the rectum is otherwise lysed of the adhesions causing obstruction and bilateral oophorectomy suffice. The decision as to whether such cauterization should be done depends upon the extent of the endometriosis in the pelvis and the age of the patient as well as her desire to bear children. Irradiation (X-ray) therapy should be reserved for those patients with known endometriosis who are poor surgical risks or who have had recurrences following conservative surgical management.

The relationship between endometriosis and carcinoma has not as yet been proved. There is considerable controversy in the literature with regard to this point.

Metastatic Malignant Growths

Although it has long been recognized that metastatic carcinoma from the cervix can produce obstruction of the rectum and colon, little mention has been made of the complete obstruction of the small bowel chiefly the ileum by such metastatic

cervical carcinoma. Reports of small bowel obstruction produced by metastasis from carcinoma of the cervix are few. Carcinoma of the cervix may spread into the parametrium and then perforate the peritoneum and spread to the serosal surface of the ileum. This may lead to annular constrictions. Direct extension to the contracting ileum may occur with invasion of the bowel wall producing complete obstruction. Although ureteral obstruction followed by uremia is a most common cause of death in carcinoma of the cervix, intestinal obstruction may be a terminal factor. It is important that this point of localization be recognized. Failure to do so with the resultant erroneous diagnosis of obstruction of the colon which is commonly made may result in a defunctionizing colostomy being performed as a palliative procedure. This is worse than useless in the presence of an obstruction of the terminal ileum.

In such cases primary resection and anastomosis or a diversionary ileocolostomy becomes the only surgical procedure feasible.

Intestinal obstruction is a common accompaniment of advanced carcinoma of the ovary. In a review of 125 established cases, Schim and Krausner found that intestinal obstruction occurred in 45.6 per cent of the cases. Of these, 27.3 per cent were found to present the signs and symptoms of intestinal obstruction which was a major factor in the patient's discomfort. An additional 18 per cent presented signs of obstruction with insignificant symptoms so that although intestinal obstruction of varying degrees of incompleteness was present there was no problem as to symptomatic treatment. In these the management did not involve the treatment of the obstruction itself. Although 45.6 per cent of the patients were obstructed to some extent, only 27.3 per cent were obstructed to a de-



FIG 114 Patient S. B. age 70 white male. Small bowel obstruction secondary to carcinoma of the colon. Note marked small bowel distention as a result of a small amount of barium given orally.



FIG 115 In this patient the Cantor tube was passed and the small bowel distention greatly reduced. Note the relative absence of distended small bowel loops. This is a postevacuation film of the colon.



FIG 116 The same patient as in Figure 115. The patient was found to have a primary carcinoma of the colon with small bowel obstruction due to metastatic deposits



FIG 117 Multiple points of small bowel obstruction due to metastatic carcinoma. Note the isolated barium filled loops

gree requiring treatment. In this group multiple levels of obstruction were found. The course of the disease is often such that surgical decompression of the obstructed intestine is not mechanically possible. Very little can be gained by decompressive procedures whether they be defunctionizing colostomy or other diversionary procedures. The patients invariably continue to complain of pain and show little or no alleviation of symptoms. In addition the life expectancy may actually be shortened by operation and furthermore instead of being made more comfortable the majority of the patients operated upon are actually made less comfortable. The consensus appears to be that surgical intervention is contra-indicated for gastrointestinal tract obstructions associated with carcinoma of the ovary except when the diagnosis is in doubt. In an exploratory, once the extent of the lesion is known it is probably better to close the abdomen without resection or diversionary procedures. If intestinal distention becomes a factor

the use of the long intestinal decompression tube is of some value in providing temporary benefits.

Meckel's Diverticulum

Johann Friedrich Meckel first described Meckel's diverticulum in 1809. Early reports were concerned with intestinal obstruction. Meckel observed that this remnant of the omphalovitaline duct could easily act as a band producing bowel obstruction. (This was probably the mechanism in the first case of intestinal obstruction due to this cause which was described by Sandifort in 1703.)

The incidence of Meckel's diverticulum ranges from 0.14 to 3 per cent. It is more common in males than in females and is generally found 3 to 100 cm from the ileocecal valve. The site seems to depend upon the amount of growth in that portion of the intestine proximal to the insertion of the vitelline duct. For this reason it is closer to the cecum in infants than it is in adults. It may be a thin fibrous band, a cystic or bulbous



FIG 118 Successful passage of Cantor tube to decompress the small bowel. Note residual distended small bowel loops and the distention in the right colon. Primary obstruction of the colon: point of obstruction the recto sigmoid.



FIG 119 Metastatic carcinoma producing small bowel obstruction. Note the marked small bowel distention.

sac with a narrow or a broad base or it may be a wide mouthed outpouching of the bowel. The most common site of origin is on the antimesenteric edge of the bowel. It may arise from the lateral surface or even the mesenteric surface of the bowel. Histologically it is a true diverticulum since its walls are identical with those of the intestine and since it contains all layers. Heterotopic tissues such as gastric, pancreatic and duodenal mucosa are found in 15 per cent of all cases.

Although most of these diverticula are asymptomatic they may become symptomatic when complications arise. Intestinal obstruction is one of the most important of these complications. Halstead reported that 6 per cent of 991 cases of intestinal obstruction reviewed were due to Meckel's diverticulum. Harkins reported Meckel's diverticulum as the cause of 2 per cent of all intussusceptions. In a review of 45 surgically treated cases of Meckel's

diverticulum at the Mayo Clinic it was found that 13 per cent were operated upon for intestinal obstruction.

Meckel's diverticulum may cause intestinal obstruction by any of the following mechanisms:

- 1 It may cause intussusception.
- 2 It may cause obstruction by being adherent to an adjacent loop of bowel with herniation of free bowel through this (adherence to abdominal wall with volvulus of loop or adherence to abdominal wall with knotting of loop of bowel about this adherent loop).
- 3 It may be found in hernial sacs causing obstruction.
- 4 Perforation of Meckel's diverticulum may result in diffuse peritonitis, local peritonitis or abscess formation. Each of these is accompanied by early paralytic ileus and possible mechanical obstruction.

The treatment of intestinal obstruction due to Meckel's diverticulum depends upon the type of obstruction produced. Simple lysis of the Meckel's



FIG 116 The same patient as in Figure 115. The patient was found to have a primary carcinoma of the colon with small bowel obstruction due to metastatic deposits.



FIG 117 Multiple points of small bowel obstruction due to metastatic carcinoma. Note the isolated barium-filled loops.

gree requiring treatment. In this group multiple levels of obstruction were found. The course of the disease is often such that surgical decompression of the obstructed intestine is not mechanically possible. Very little can be gained by decompressive procedures whether they be defunctionizing colostomy or other diversionary procedures. The patients invariably continue to complain of pain and show little or no alleviation of symptoms. In addition the life expectancy may actually be shortened by operation and furthermore instead of being made more comfortable the majority of the patients operated upon are actually made less comfortable. The consensus appears to be that surgical intervention is contra-indicated for gastro-intestinal tract obstructions associated with carcinoma of the ovary, except when the diagnosis is in doubt. In an exploratory, once the extent of the lesion is known, it is probably better to close the abdomen without resection or diversionary procedures. If intestinal distention becomes a factor

the use of the long intestinal decompression tube is of some value in providing temporary benefits.

Meckel's Diverticulum

Johann Friedrich Meckel first described Meckel's diverticulum in 1809. Early reports were concerned with intestinal obstruction. Meckel observed that this remnant of the omphalovitteline duct could easily act as a band producing bowel obstruction. (This was probably the mechanism in the first case of intestinal obstruction due to this cause which was described by Sandifort in 1793.)

The incidence of Meckel's diverticulum ranges from 0.14 to 3 per cent. It is more common in males than in females and is generally found 3 to 100 cm from the ileocecal valve. The site seems to depend upon the amount of growth in that portion of the intestine proximal to the insertion of the vitelline duct. For this reason it is closer to the cecum in infants than it is in adults. It may be a thin fibrous band, a cystic or bulbous



FIG 118 Successful passage of Cantor tube to decompress the small bowel. Note residual distended small bowel loops and the distention in the right colon. Primary obstruction of the colon: point of obstruction the recto-sigmoid.



FIG 119 Metastatic carcinoma producing small bowel obstruction. Note the marked small bowel distention.

with a narrow or a broad base or it may be a wide mouthed outpouching of the bowel. The most common site of origin is on the antimesenteric edge of the bowel. It may arise from the lateral surface or even the mesenteric surface of the bowel. Histologically it is a true diverticulum since its walls are identical with those of the intestine and since it contains all layers. Heterotopic tissues such as gastric, pancreatic and duodenal mucosa are found in 15 per cent of all cases.

Although most of these diverticula are asymptomatic they may become symptomatic when complications arise. Intestinal obstruction is one of the most important of these complications. Halstead reported that 6 per cent of 991 cases of intestinal obstruction reviewed were due to Meckel's diverticulum. Harkins reported Meckel's diverticulum as the cause of 2 per cent of all intussusceptions. In a review of 45 surgically treated cases of Meckel's

diverticulum at the Mayo Clinic it was found that 13 per cent were operated upon for intestinal obstruction.

Meckel's diverticulum may cause intestinal obstruction by any of the following mechanisms:

- 1 It may cause intussusception.
- 2 It may cause obstruction by being adherent to an adjacent loop of bowel with herniation of free bowel through this (adherence to abdominal wall with volvulus of loop or adherence to abdominal wall with knotting of loop of bowel about this adherent loop).
- 3 It may be found in hernial sacs causing obstruction.
- 4 Perforation of Meckel's diverticulum may result in diffuse peritonitis, local peritonitis or abscess formation. Each of these is accompanied by early paralytic ileus and possible mechanical obstruction.

The treatment of intestinal obstruction due to Meckel's diverticulum depends upon the type of obstruction produced. Simple lysis of the Meckel's

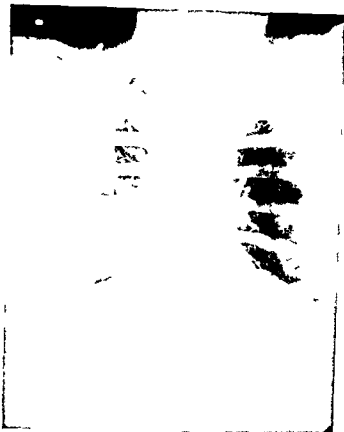


FIG 120 The same patient as in Figure 119. The value of radiographs of the chest is amply demonstrated: metastatic malignant deposits are quite apparent on this film, thus making the diagnosis of the etiology of the small bowel obstruction relatively certain.



FIG 121 Meckel's diverticulum with adhesions across the ileum producing a tremendous small bowel obstruction. The hugely distended bowel noted on this radiograph is a loop of terminal ileum lying between the Meckel's diverticulum and an obstruction due to an adhesion of the terminal ileum distal to this point. The hugely distended ileum came to lie in the left upper quadrant as noted on the film.

brand at its point of attachment to the bowel with inversion of this point may be all that is required. If the bowel is strangulated, primary resection and anastomosis may be necessary. In some cases the diverticulum may be transected at its base and inverted into the bowel in two layers, the distal portion then being removed. At times bowel resection is imperative to adequately remove the diverticulum. In those cases associated with intussusception the intussusceptum should be milked out, and then the diverticulum removed. In all such cases the diverticulum should be transected at its base in the long axis of the bowel and the opening closed transversely to avoid narrowing of the bowel lumen. If the intussusceptum has become strangulated or cannot be milked out, primary resection of the entire mass and end-to-end anastomosis may be necessary. If Meckel's diverticulum has resulted in a perforation with peritonitis or abscess formation, the treatment is the same as

though one were treating a ruptured appendix. The use of the long intestinal decompression tube to treat the associated paralytic ileus is of inestimable value.

Intussusception

Intussusception in older children and adults differs in many respects from the condition seen in infants. The incidence of this disorder in adults has been variously reported from 0.003 to 0.02 per cent of all hospital admissions. Adults are believed to account for from 5 to 10 per cent of all intussusceptions, the remaining 90 to 95 per cent being found in infants under two years of age. The operative treatment of intussusception in adults was reported in the literature as early as 1700. John Hunter gave the first complete description in 1733.

Brayton and Norris reviewed the literature and

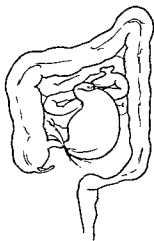


FIG. 122 The same patient as shown in Figure 121 with a Koslow tube in the stomach. Note that the tremendously distended bowel is not stomach since the stomach has previously been emptied by the Koslow tube. Note the tremendously distended small bowel loop lying in mid abdomen. The diagnosis was not made prior to surgery.

reported 745 cases of intussusception in adults. They noted that whereas in infants the disease is primary in 95 per cent of cases, no cause being found for the intussusception in adults, the opposite is true. The causes of intussusception in the

745 adults reviewed by Brayton and Norris are as follows:

1 Primary	167 cases (22%)
2 Benign tumors	246 cases (33%)
3 Malignant tumors	174 cases (21%)
4 Meckel's diverticulum	45 cases (6%)
5 Stomal	33 cases (4.4%)
6 Prolapse of gastric mucosa	27 cases (3.6%)
7 Chronic ulcerations	16 cases (2.1%)
8 Adhesions	5 cases (0.6%)
9 Aberrant pancreas	3 cases (0.4%)
10 Trauma	3 cases (0.4%)
11 Foreign body (including Miller Abbott tube)	3 cases (0.4%)
12 Miscellaneous	2 cases (0.2%)
13 Secondary intussusceptions (cause not given)	41 cases (5.5%)

In a review of adult intussusception at the Cleveland Clinic it was found that of 430,000 admissions to the hospital in one period of time 15 cases of adult intussusception were recorded. Only one patient of the 15 presented a primary type of intussusception. The remaining 14 were secondary to tumors of the bowel. Of these 14 tumors of the bowel 8 were found to be malignant (6 carcinoma and 2 sarcoma) and 6 were found to be benign. This varies considerably with the incidence for large groups of patients in the series reported by Brayton and Norris and Donhauser and Kelly.

Intussusception may be found at any age but its incidence decreases rapidly after early adult life. This is readily apparent in the statistical review by Donhauser and Kelly. In this group the following age distribution was found: 15 to 30 years—225 cases; 31 to 45 years—201 cases; 46 to 60 years—147 cases; 61 to 80 years—65 cases; and 81 to 90 years—2 cases.

Intussusception in adults appears to be more common in the Orient than it is in the Occident. In a review of 28 cases of intussusception treated at the Lester Chinese Hospital by Ma An and Shen, more than 50 per cent of the patients were over 10 years of age. This is in contradistinction

to the incidence of intussusception above the age of 10 in the Caucasus race

Contrary to the findings in infants in whom intussusception begins as an acute intestinal obstruction intussusception in the adult generally is chronic and intermittent in nature. The patient usually reports recurring small bowel obstruction which finally becomes complete. The four cardinal findings associated with intussusception in adults are (1) abdominal pain of a cramping nature (2) vomiting (3) palpable mass in the abdomen and (4) passage of a bloody stool. Any one of these findings is considered to be suggestive of intra abdominal pathology. All four findings are rarely found in any one case. Although the diagnosis may be very difficult to make it can be made if intussusception is suspected.

All the symptoms noted with intussusception are those of intestinal obstruction in general. The classic symptoms of intussusception are caused by the repeated hyperperistaltic efforts to empty the bowel. This produces a cramping pain. Obstipation is the result of intestinal obstruction after the bowel distal to the obstructive process has been emptied. The passage of a bloody mucus per rectum is the result of extravasation of blood and lymph through the walls of the obstructed channel. Vomiting comes on late in low ileal obstructions whereas in jejunal intussusception it may come on relatively early. Borborygmus may be readily audible in association with the colicky pain. There is generally little or no tenderness because the incarcerated intussusceptum lies within a sheath of normal bowel whose serosa is not inflamed. An abdominal mass is found in less than 50 per cent of the reported cases. A diagnosis of mechanical intestinal obstruction can readily be made and appropriate surgical treatment instituted. If intussusception is found in an adult a careful search should be made for the tumor mass which so often initiates the process.

Schatsky outlined the radiologic features of intussusception and pointed out that the coiled spring appearance is pathognomonic if found. It is produced by barium suspension insinuating itself between the intussusceptum and intussusciens. Other radiologic features helpful in diagnosis are absence of normal gas pattern on sur-

vey film, and a narrow column of barium in the intussusception suggesting Kantor's string sign of regional ileitis.

The gross pathologic changes associated with intussusception are caused by the constricting action of the neck of the intussusciens. First the venous and lymphatic channels become obstructed. This produces edema and extravasation of the fluid. The arterial blood supply is occluded last so that gangrene is a late feature of this type of obstruction. In such cases, the intussusceptum becomes strangulated. During this period of time the lumen of the bowel may become closed as a result of the presence of the intussusceptum. Complete intestinal obstruction results and the signs and symptoms characterizing this abdominal emergency become unmistakably manifest.

The most common error in cases of intussusception in young adults is to diagnose the disorder as a ruptured appendix with localized abscess formation. There is often sufficient similarity in physical findings to lead the unwary astray. The important distinguishing features in differentiating between these dissimilar conditions are

1. In intussusception the acute attack is often preceded by blood in the stool.

2. The obstructive process is characterized by borborygmus and colicky pain. This does not occur with an appendical abscess. Here paralytic ileus is the usual accompaniment.

Intussusception may occur anywhere along the gastro intestinal tract below the esophagus. It has been classified as gastrogastic, gastroduodenal, enteric, ileocolic, ileocecal, appendiceocolic, colic and stomal. In the adult the majority of the intussusceptions are enteric involving either the jejunum or ileum. These may be classified as jejunojejunal, jejuno ileal or ileo ileal. The intussusception usually occurs in the direction of normal peristalsis. An occasional case of retrograde intussusception is reported as a medical oddity. The intussusceptum may extend a long distance into the intussusciens. Cases have been reported in which the intussusceptum was found protruding from the anus.

Intussusception of the Appendix. Inversion and intussusception of the appendix with protrusion of the cecum and appendix through the anus

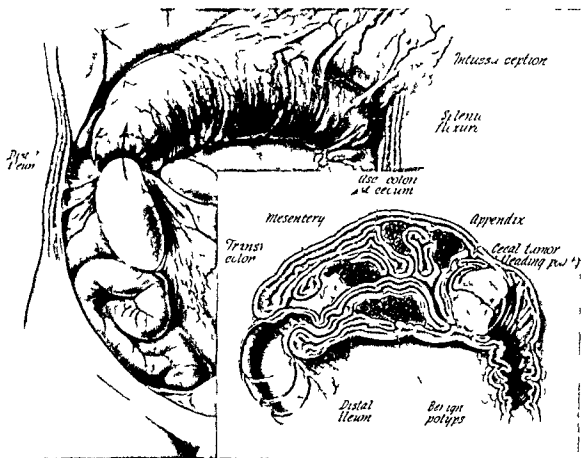


FIG 123 Tumor of the cecum causing intussusception. Notice the distance the intussusceptum has traveled along the colon in this illustration. The head of the intussusceptum has come to lie at the splenic flexure.

is a rare occurrence. Dunavant and Wilson reported an 8 month old child in whom there was complete inversion of the appendix with protrusion of the appendix and cecum through the anus. McSwain reviewed the literature in 1941 and collected 77 cases of intussusception of the appendix. He found six cases in which the appendix extended far enough distally to be palpable per rectum. Russell and Gravelle reported a case of intussusception following appendectomy. Seven similar previously reported cases were found. Interestingly enough in six of the cases the stump of the appendix was inverted and in one case it was not. In the case reported by Russell and Gravelle although the stump of the appendix was inverted it was found not to be the nidus for the intussusception. It is not uncommon in cases of this type for the intussusceptum to be necrotic and require resection. These cases of intussusception

following appendectomy were found to occur anywhere from two or three days to as late as one week after surgery. A specific diagnosis is difficult and is rarely made.

Intussusception Caused by Foreign Bodies

Jejunal and ileal intussusceptions have been reported as a result of the presence of foreign bodies. This is one of the possible complications in the use of the long intestinal decompression tube. Smith and Van Beuren report a case in which intussusception resulted from the balloon of the Miller Abbott tube. Similar isolated reports were made by Harris, Howard and Warren and Cattell as well as Dunn and Shearburn. The same physiologic forces which produce intussusception of the small bowel at the site of any space occupying lesion are responsible for intussusception in this case. The bulky end of the tube is physically analogous to a space occupying bowel lesion. Since

the intussusceptions produced were not of the reversed variety they did not result from the withdrawal of the tube

The treatment of all types of adult intussusception is surgical. Conservative attempts at reduction of the intussusception must never be used in this age group. In addition to correction of the intussusception the etiologic agent producing it should be looked for and removed. This may necessitate bowel resection and end to end anastomosis. In all other instances if the intussusceptum after being milked out is found to be viable nothing further is required. In those cases in which strangulation of the intussusceptum has occurred or in which the viability of the bowel is questionable a primary resection of this portion of the bowel is safest.

Volvulus

Volvulus develops due to fixation of one portion of the bowel with a freedom of motion in the adjacent bowel. The combination of partial fixation of one segment and normal mobility of another allows rotation of the intestine around the base of the mesentery with the formation of a volvulus. Once the primary loop has formed further torsion may occur.

There are many causes for the development of volvulus of the small intestine. All cases of volvulus when classified as to cause may be divided into two large groups: (1) congenital and (2) acquired. Of these the majority of small bowel volvulus in adults occurs as a result of acquired causes.

Most cases of small bowel volvulus in adults are a direct result of postoperative or inflammatory adhesions. These may be secondary to an inflammatory process in a specific organ such as appendicitis or Meckel's diverticulitis with fixation of the inflamed organ to the parietal peritoneum to another fixed organ such as the uterus or to the posterior surface of the broad ligament. Torsion may develop in the immediate postoperative period or at some later time. Rough manipulation of the bowel in returning it to the peritoneal cavity may be a responsible factor. A loop of bowel may become adherent to the anterior abdominal wall. Torsion of a loop fixed in this fashion is not un-



FIG. 124. Intestinal obstruction caused by volvulus of a loop of ileum adherent to the posterior surface of the uterus and to a left tubo-ovarian abscess. Note the fluid level in the obstructed bowel.

common. A loop of bowel may encircle a part of bowel so fixed with a resultant knotting and obstruction. The greater omentum may be adherent to the anterior abdominal wall or to the pelvic organs. A twisting of a loop of bowel around this may occur. Fixation of a loop of bowel in an incarcerated hernia increases the tendency of the proximal portion to twist.

Dietary factors are considered to be common causes for small bowel volvulus. Kallio reported that there was a relatively high incidence of volvulus among the Finns in Finland but not among Finns in Minnesota. This was explained by the difference in food eaten in the two countries. The food in Finland is voluminous in quantity, badly prepared and often low in caloric value. The encourages intestinal distention which Kallio believed to be associated with certain congenital factors in causing volvulus. Sweet reported that volvulus of the small bowel was more frequent in Russia. Fin-



FIG. 1. Lateral view of the patient in Figure 124. Note the small bowel distention and fluid level.

land Poland and Scandinavia because of the coarse food eaten in those countries and the resultant tendency toward intestinal distention. In some instances active exercise and hyperperistalsis caused by cathartics may be contributing factors. In eastern European areas the percentage of volvulus has been reported as ranging from 30 to 40 per cent of all cases of intestinal obstruction. The diet of eastern European peoples consisting chiefly of coarse foods with a high cellulose content predisposes to intestinal overloading and distention of the bowel by fermentation and putrefaction. Lambbridge reported that in Kenya volvulus of the small bowel is the commonest cause of acute intestinal obstruction, being responsible for almost 50 per cent of all cases in the natives. This has been directly attributed to the dietary habits. It is the custom of the African native to start the day by drinking up to two pints of maize meal gruel in the early morning and again at midday and then taking nothing solid until the evening meal. The sudden distention of the small in-

testine by thick liquid causes an overloading of one or two loops which during exertion may become displaced and initiate a volvulus. The resultant hyperperistalsis then completes the process. Evidence that such large meals may be responsible for the onset of volvulus is further suggested by the findings of McWatters who reported on intestinal obstruction in India. It is the dietary habit of the Mohammedans in India during Ramadan to fast from sunrise to sunset and then feast. Just as in the case of the African native the sudden gross overloading of the bowel is believed to be the responsible factor in causing obstruction.

Among the uncommon causes of volvulus are the following:

- 1 A tumor within the bowel consisting of a coarcted worm. The bowel may be fixed by the weight of the tumor mass causing it to twist.

- 2 Large gallstones may so weight the bowel as to initiate volvulus.

- 3 Volvulus may be caused by mesenteric cysts or enterogenous cysts.

- 4 Occasionally there may be an intertwining of two loops of bowel. Entanglements of this type occur most often between the sigmoid colon and a loop of terminal ileum and are rarest between the cecum and a loop of small bowel. Paul reported a case in which the patient presented an unduly long loop of sigmoid colon with a very narrow base. A loop of terminal ileum swept around the sigmoid colon at its base from right to left and passed under the arch of the small bowel producing intestinal obstruction of the small bowel of the volvulus type.

- 5 An association of volvulus with intussusception although uncommon is by no means rare. Quilliam reported cases of this type.

- 6 Lead poisoning has been reported as an uncommon cause of volvulus of the small bowel. Berger and Lundberg reported a group of five cases of volvulus precipitated by lead poisoning, four of which involved the sigmoid colon and one of which involved the small bowel. All the patients had worked in lead plants anywhere from eight months to seven years. At the time of hospitalization they presented pronounced evidence of lead absorption as well as prior symptoms con-

sistent with gastro intestinal plumbism. During an episode of gastro intestinal lead poisoning there is an intestinal hyperactivity consisting of uncoordinating muscular spasm of the bowel. This gives rise to the severe colic associated with lead poisoning and in addition may mimic intestinal obstruction. The colicky abdominal pain, nausea, vomiting, obstipation and even abdominal tenderness may suggest bowel obstruction. Distention is not mentioned as a prominent feature of pure lead poisoning, although Berger and Lundberg did find distention in several instances of uncomplicated lead colic. They demonstrated small bowel and large bowel distention on physical examination as well as radiologically in several cases. The intra venous administration of calcium is of great value in the differential diagnosis. The colicky pain of pure lead poisoning is relieved by the administration of calcium, whereas in the superimposed organic intestinal obstruction the colicky pain persists.

Among the congenital causes of small bowel volvulus are nonrotation, reversed rotation or malrotation of the gastro intestinal tract. A Meckel's diverticulum may initiate the process. Internal hernias are not uncommonly associated with volvulus. A rather uncommon cause for congenital volvulus is the presence of an abnormally placed appendix. Among the group of small bowel obstructions caused by congenital factors, a true midgut volvulus is fairly common. As opposed to the small bowel volvulus in which only the small intestine is twisted on its mesenteric axis, the true midgut volvulus is one in which the right colon as well as the entire small bowel and even the duodenum may be twisted upon a common mesenteric axis. If there is a complete nonrotation of the intestinal tract with failure of secondary fixation of the mesentery, the long common ileocecal mesentery associated with a short dorsal mesenteric attachment is found. In such instances, volvulus would involve the right colon as well as the small bowel, producing a true midgut volvulus. If partial rotation occurred, the cecum may have become arrested in the right upper quadrant. A narrow mesenteric pedicle from the cecum to the closely placed duodenojejunal junction permits volvulus of the small bowel alone. Various de-

grees of malrotation and associated deficient intestinal fixations may also give rise to this condition. The rarest anomaly of the second stage of bowel rotation is retroposition of the transverse colon. This is a congenital disturbance in which the transverse colon comes to lie behind the superior mesenteric vessels and the third portion of the duodenum lies in front of them. Retrodisplacement of the transverse colon in this position has been reported complicated by ileocecal volvulus. It has been noted that different types of retroposition of the transverse colon occur. The different types depend upon the amount of fixation of the mesenteric root of the small bowel. If the mesenteric root is wide, it acquires a normal secondary attachment and so must cross the transverse colon which runs through a tunnel in the anterior wall of the superior mesenteric vessels, to appear. If the root of the mesentery is narrow, the mesentery stops above the level of the transverse colon which therefore does not run through a tunnel. It then remains free behind the small bowel and the whole midgut is suspended from the original primitive mesentery and is very likely to undergo volvulus.

Lee reported a case of volvulus of the terminal ileum, cecum, ascending colon and the portion of the transverse colon to the right of the mesenteric vessels through a congenital hole in the mesentery of the small bowel. An abnormally placed appendix is an uncommon cause of volvulus. Cohn and Felmus reported an uncommon case in which the appendix was found to come off of a highly placed cecum. It then passed around the root of the mesentery on the inner side and became adherent by its tip to the brim of the pelvis on the other side of the mesentery. Volvulus of loops of bowel around this abnormally placed appendix resulted.

Midgut volvulus appears to be more common in males than in females. This is attributed in part to the effect of vigorous exercise on the production of volvulus.

Volvulus may occur in any age group. The youngest patients reported are the newborn who present volvulus as a congenital anomaly. The oldest patients have been in their eighties. The great

est incidence of volvulus is in the 10 year period between 30 and 40 years of age

The pathologic changes of volvulus are similar to those found in intestinal obstruction associated with interference to the circulation of the obstructed loop of bowel. There may be complete or incomplete obstruction. There may or may not be interference with the blood supply to the bowel. When the twist is tight strangulation of the involved loop of bowel is common. The obstruction may be acute, subacute or chronic depending upon the tightness of the twist as well as the degree of twisting. The amount of intestinal rotation is variable from twists through an arc of 90 degrees to those of 360 degrees or more. The tightness of the twist is as important as the number of turns in the production of strangulation of the bowel so obstructed. The circulatory disturbance may vary from slight obstruction of the venous return to complete occlusion of the arterial and venous blood supply. The ileum is involved in the majority of cases. Volvulus appears approximately eight times as often here as in the jejunum.

The symptomatology associated with volvulus of the small bowel is that of acute small bowel obstruction. There are no pathognomonic symptoms indicative of volvulus. The clinical picture of intestinal obstruction becomes evident early in the course of the process when the twist is sufficient to obstruct the bowel. Colicky pain of sudden onset is the first symptom complained of. This continues until the process is relieved or until strangulation of the bowel occurs. In this latter event the pain becomes dull and continuous and abdominal tenderness appears. Nausea, vomiting and abdominal distention appear later in the course of the obstruction if the terminal ileum is involved. The temperature and pulse are not elevated unless strangulation is present. Then both may be elevated or the pulse alone may show an elevation. The white blood count is usually normal unless strangulation occurs. Then an absolute or relative leukocytosis is common. A specific diagnosis of volvulus is difficult because of its lack of characteristics, the best that usually can be achieved is a diagnosis of acute intestinal obstruction.

The treatment for volvulus is early surgical intervention. Intestinal intubation except as an ad-

junct to immediate surgery is of no value and may be dangerous. The tendency for strangulation to occur early is sufficient indication for prompt surgical treatment. If strangulation has not occurred simple detorsion of the twisted loop is sufficient. If strangulation of the bowel has occurred or if the viability of the bowel is questionable primary resection and end-to-end anastomosis is the treatment of choice. If volvulus is associated with congenital anomalies such as retrodisplacement or nonrotation of the colon after detorsion of the obstructed loop no attempt should be made to correct the abnormality.

The mortality rate associated with volvulus is high with rates as high as 46 per cent reported. This is chiefly due to the interference with the blood supply. Gangrene of the obstructed loop is all too common in such cases. Early surgery is the only method by which this mortality rate can be reduced. The indiscriminate use of the long intestinal decompression tube and the institution of the watchful waiting policy are undoubtedly responsible for many of the deaths.

Diverticulitis

The first jejunal diverticulum was described by Cooper in 1807 as an autopsy finding. Osler was the first to describe jejunal diverticulum in the American literature in 1881. This too was an autopsy finding. Gordinier and Sampson in 1906 first reported jejunal diverticulum found during the course of surgical procedure for intestinal obstruction. About 300 cases of this type have been reported to date.

Jejunal diverticula are the rarest of outpouchings of the gastro-intestinal tract. They are represented by a herniation of the mucous membrane of the bowel along the point of blood vessel entry. This usually occurs along the mesenteric side of the bowel although in a small percentage of cases it may occur on the antimesenteric edge. At this point of vessel entry the bowel wall is considered to be deficient. As a result any increase in intraluminal pressure at this point may create a bulge of the mucous membrane between the blood vessel and the adjacent muscularis of the wall. As the intraluminal pressure is constantly applied an



FIG 126 Intestinal obstruction caused by jejunal diverticulum with ulcer and perforation Magnification $\times 150$

resistance is unable to oppose it the diverticulum becomes permanent

These lesions are usually asymptomatic and only produce symptoms when complications occur. Of these acute and chronic obstruction of the bowel is most common. This may be caused by different mechanisms: (1) pressure of the filled diverticulum (2) adhesions due to diverticulitis (3) enteroliths forming within the diverticulum causing pressure obstruction (4) perforation of the diverticulum causing general peritonitis, local peritonitis or localized abscess with reflex paralytic ileus. Many of these cases go on to mechanical obstruction.

The management of intestinal obstruction due to jejunal diverticulum is surgical. Resection of the diverticulum may adequately correct the problem. In some cases resection of the loop of jejunum is indicated. In those cases in which ileus or obstruction is associated with a perforation of a diverticulum conservative treatment such as one would institute in the management of an appendiceal abscess is the treatment of choice. The use of the long intestinal decompression tube anti-

otics electrolytes and water is of great value in the treatment of obstructions of this inflammatory distention group.

Mesenteric Vascular Occlusive Disease

The all inclusive 'mesenteric vascular occlusive disease' is used to cover all disorders to the blood supply of the small bowel. Vascular occlusions regardless of etiology, result in anoxia of the bowel supplied by the occluded vessel. When the blood supply is grossly interfered with death of the bowel results. Intestinal obstruction occurs when there is any interference with the proper nutrition of the bowel wall. This is variable in degree ranging from disturbances in peristaltic activity by a disorganized propulsive mechanism to the extreme bowel obstruction associated with gangrene of the bowel.

Three types of mesenteric vascular occlusive disorders are recognizable: (1) a primary type in which the intestinal obstruction is secondary to vascular occlusion, (2) a secondary type in which the intestinal obstruction is the initial disorder and the vascular occlusion is the result of strangulation by compression of the mesentery of the bowel or twisting of its vascular pedicle and (3) the rare polyarteritis nodosa of the mesenteric vessels with infarction of the small bowel.

The primary type of mesenteric vascular disease was classified as mesenteric thrombosis or embolism in older publications. The most common factors responsible for this type of occlusion are: (1) cardiac disease such as auricular fibrillation, coronary disease and valvular heart disease (2) thromboembolic diseases such as acute and subacute bacterial endocarditis (3) blood dyscrasias (4) aortic calcifications and vegetations (5) appendicitis with pylephlebitis (6) cirrhosis of the liver (7) carcinoma of the pancreas and pancreatitis with compression of the superior mesenteric artery (8) abdominal trauma. In some cases no apparent cause is found.

The primary type of mesenteric vascular occlusive disease is uncommon. In a series of 30,000 admissions to Charity Hospital, Boyce and McFetridge reported 15 cases of this disease. Schiff and Acker reviewed 22,622 major surgical procedures over a 5 year period. In this group only

12 cases of mesenteric occlusion were found. Only eight were operated upon. This abdominal catastrophe was first noted by Trendelenburg in 1843 & is credited with the first description of the pathology involved.

The primary type of mesenteric vascular occlusion may be further classified for purposes of prognosis into different groups depending upon whether the obstruction is arterial or venous or both. An arterial mesenteric occlusion may be either thrombotic or embolic. The venous type of occlusion is generally thrombotic. There is considerable difference in the course of the disease depending upon whether the obstruction is venous or arterial. Laufenberg suggested that the following additional factors are of great importance in determining the course of the disease: (1) the duration of circulatory obstruction (2) the amount of blood extravasated into the lumen of the bowel (3) metabolic activity of the tissues at the time of vascular occlusion (4) the presence of a closed loop (5) length of bowel segment (6) number of vessels occluded (7) the speed of onset of the occlusion (8) the presence of vasospasm.

In arteriovascular occlusive disease, arteriovascular spasm is a prominent feature of the process. At an early stage the bowel appears bunched. This creates a tissue anoxia as a result of which the spasm of the bowel relaxes and the bowel becomes cyanotic. It has been amply demonstrated that in this type of occlusion if the arterial occlusion can be released within two to six hours the viability of the bowel will be restored. The secondary changes in the bowel become reversed. Carucci studied the mesenteric vessels in 20 specially selected necropsy specimens in an attempt to determine whether there was a recognizable primary sclerosis. It was found that although the aorta demonstrated evidences of a marked sclerosis in approximately 20 per cent of the cases there was no evidence of vascular sclerosis in the mesenteric vessels beyond the short origin of the superior mesenteric artery. This observation becomes significant in view of the fact that many of the specimens ranged in age from 70 to 80 years and in these marked sclerosis was found in the arteries in other portions of the body. In contradistinction to this are the isolated case reports in which mesenteric thrombosis with

infarction occurred following resection of the left colon with ligation of the inferior mesenteric artery. Autopsy studies in several cases demonstrated atheromatous plaques shortly beyond the origin of the superior mesenteric artery so reducing the caliber of this vessel as to cause thrombosis.

Shaw and Green reported massive mesenteric infarction after inferior mesenteric artery ligation in resection of the colon for carcinoma. They believed that the patient had gradually suffered an occlusion of the superior mesenteric artery and had become dependent on the collateral channels from the inferior mesenteric artery and the celiac axis for circulation to the small bowel. High ligation of the inferior mesenteric artery in this case left only the collateral circulation from the celiac axis. As a result with the small residual flow through the markedly narrowed superior mesenteric artery the stage was set for the development of thrombosis and infarction. Moore, in a review of mesenteric vascular occlusion called attention to the known reported cases in which gradual arteriosclerotic occlusion of the superior mesenteric artery had occurred with a development of the collateral circulation to the bowel by means of such cases there was little or no organic change in the bowel and only an occasional functional disturbance. It has been suggested that this situation may exist asymptotically more frequently than is recognized.

Carucci studying 20 cadavers from the point of view of the mesenteric circulation found that in addition to the collateral circulation reported the right colic and middle colic arteries may be found in a rare case to arise from the left colic branch of the inferior mesenteric artery. An additional case has been reported in which one single main trunk gave rise to the various branches of both the superior and the inferior mesenteric artery. In a case of this type it should be obvious that ligation of the inferior mesenteric would undoubtedly result in infarction of the small bowel. Although these abnormalities in blood supply are uncommon and may even be considered to be rare nevertheless they can occur. For this reason in cases in which ligation of the inferior mesenteric artery is to be

used prior to resection of the left colon a careful examination should be made to note the arterial pattern before such ligation is performed

Venous mesenteric occlusion presents a far more serious problem. In this type of case a hemorrhagic infarction of the involved segment of bowel occurs. The venous occlusion must be released within the first two hours if a damaged bowel is to be avoided. Passive congestion and edema occur very rapidly with venous thrombosis. Retrograde thrombosis initiated by the initial obstruction is common. The danger of progression of the thrombosis after resection for mesenteric venous occlusion must be remembered. A breakdown of the anastomotic site will occur unless sufficient bowel is removed to insure an adequate arterial and venous blood supply. This type of occlusive disease is commonly found with strangulating bowel obstructions caused by compression of the mesenteric pedicle of the obstructed bowel.

Mesenteric vascular occlusions have been reported as a result of polyarteritis nodosa. Both arteries and veins may be attacked by this disease. The histopathologic changes reported are characteristic of an inflammatory reaction which is progressive and pursues a definite pattern occurs around the vessels. In the early stages of the disease there is an acute inflammatory cell exudate composed chiefly of polymorphonuclear cells, eosinophils, and lymphocytes noted in and around the small and medium sized blood vessels. Necrosis of the media may be noted. As the disease progresses necrosis of the vessel wall and thrombosis of the lumen follow. The end result is replacement of the cellular exudate by fibroblasts which finally obliterate the vessel. Judging from the available literature polyarteritis nodosa is a rare lesion. Infarction of the bowel is confined to the occluded vascular area. The etiology of this disease is unknown despite the volume of clinical and experimental investigation. The most promising concept at present is that of Gruber who suggested that the disease was due to hypersensitivity. Combined arterial and venous thrombosis is almost as common as pure arterial occlusion.

Mesenteric vascular occlusions occur at any age but are found most commonly between the ages



FIG. 127. Periarteritis nodosa. Notice the marked small bowel distention.

of 30 and 60 years. Males are afflicted three times as often as females.

Mesenteric occlusive disease may be of two types: (1) acute and (2) subacute or chronic. Those classified in the acute group constitute major surgical emergencies. The onset of symptoms ranges from one to six days with an average of three days. Many patients die of shock within a short period of time while others die in several days of such causes as blood loss within the bowel, lumen perforation, and overwhelming toxemia. The predominant symptoms in this acute group are the onset of severe cramping abdominal pain associated with some degree of shock. The latter may vary from mild hypotension to a severe degree of irreversible shock. The pulse is usually rapid and there may be a bloody diarrhea. Early in the course of the process hyperperistalsis is very evident. There is a marked intestinal activity at this time. In patients examined during this period there may be marked disparity between the paucity of physical abdominal findings and the clinical picture of a seriously ill patient. In less



FIG. 128. Ra diograph of chest indicative of periarteritis nodosa. This is the same patient as in Figure 127. The correlation of the radiographic chest findings with the abdominal findings is typical of periarteritis nodosa.

severe cases of this type pulse temperature and blood pressure are within normal limits. When seen later in the course of the disease the peristaltic activity may be greatly decreased and some degree of abdominal distention may appear. This is not a reliable sign of mesenteric vascular occlusion. The most important point in the diagnosis of occlusive mesenteric disease is the disparity between the severity of pain experienced and the relatively benign signs and abdominal findings presented.

In many cases of occlusive vascular disease the symptoms may extend over a period of a week or even a month with many remissions and relapses. These are classified as sub-acute or chronic. These patients complain of vague abdominal pain and may present symptoms suggestive of partial intestinal obstruction. Few physical findings may be noted. In these cases the onset of the illness may be so mild and extend over so long a period of time that diagnosis is difficult.

This disease should be treated surgically as soon

as a diagnosis is made. (It is interesting to note that in few of the cases operated upon has the pre-operative diagnosis been correct.) In some cases spontaneous cure was reported when small radicles of vessel were obstructed. This was due to the rich collateral blood supply. Although immediate surgery is the recommended method of treatment for these lesions an occasional elderly patient in poor condition may be saved by massive doses of antibiotics to keep the bowel sterile and the use of the long intestinal decompression tube to combat distention. Heparin and Dicumarol may be used in such cases. Some otherwise hopeless patients can be salvaged by this technique. In one such patient surgery after recovery demonstrated a contracted stenosed bowel as a result of healing of the infarcted area. Aside from these occasional cases the treatment must be surgery performed at the earliest possible moment. The decision as to how much bowel to resect or whether to resect any must be made at the time of surgery. Ross and Reed reported recovery in a patient in whom occlusion of the superior mesenteric artery had occurred. Such occurrences must be uncommon however.

A mortality rate from 60 to 90 per cent is being reported in the management of the enteric occlusive vascular disease.

Anomalies in Development

Abnormalities of intestinal rotation and mesenteric fixation cause more obstructive symptoms in adults than is realized. An uncommon cause of intestinal obstruction in the human is midgut volvulus due to nonrotation of the human intestine. (This disorder is common in the dog.) The colon comes to lie in the left side of the abdomen with the small bowel to its right. The terminal ileum enters the cecum on the right side and laterally. In some cases the bowel may be in a stage of incomplete rotation so that the cecum comes to rest in the epigastrium being fixed in this position by fibrous bandlike attachments to the under surface of the liver and the lateral abdominal wall. As a result the small bowel hangs suspended by a small pedicle from the site of origin of the superior mesenteric artery. Volvulus may then occur with this pedicle as an axis. The lack of fixation as occurred

with the failure of the second or third stage of intestinal rotation or occurring in the presence of a normally rotated intestine, may cause intestinal obstruction. The obstruction in such cases is produced by the abnormal mobility which permits twisting of a segment of the whole bowel. The lack of fixation is compatible with digestive good health but when symptoms do occur they may be the result of volvulus, intussusception or occasionally internal herniation with obstruction. The most common type of failure of fixation of the mesentery results when there is a failure of the third stage of intestinal rotation. In such cases the cecum and proximal segment of the colon are left with varying degrees of mobility. The anomalous fixation may then produce obstruction by two different mechanisms: (1) volvulus of the entire mass of small bowel may occur because of a failure of fixation of the small bowel; (2) mesenteric congenital bands may result which cause intermittent and at times complete obstruction. Gardner and Hart reported two cases of their own and reviewed 98 cases from the literature in which the entire small bowel had undergone volvulus due to a twisting about the mesenteric pedicle. A mobile cecum may serve as an axis around which a section of small bowel may become coiled, thus causing volvulus.

Allergy

Allergy must be kept in mind as a possible cause of gastro intestinal obstruction. Bednfield reported that Spigelius and Riola in the 1640s described narrowings in the bowel of cadavers which they termed "angry contractures" and which they thought were due to enterospasm. In recent years it has been demonstrated that allergy is capable of causing severe and serious intestinal disturbances. Fogart reported a case in which the lumen of the jejunum was almost completely occluded by edema of its wall as a result of hypersensitivity. There is little doubt that such a reaction can occur with allergy. The vascular reactions to allergy are capillary dilatation with increased permeability and exudation as well as spasm of the arterioles. These reactions vary greatly in degree. Gallison reported a case in which the intestinal obstruction was proved to be due to allergy.

It might be advisable to look for a history of allergy in any case of intestinal obstruction where the etiology is not definite. Several hours spent with a trial of intramuscular antihistamines in selected cases might prevent an unnecessary operation. A failure of response to such antihistamine therapy within two hours must be considered presumptive evidence that allergy does not play a part. Whoever makes a diagnosis of intestinal obstruction due to allergy, treads on dangerous ground if by so doing proper surgical treatment is delayed.

Functional Small Bowel Obstruction

Bonney described a functional intestinal obstruction which he called paralytic ileus. This is considered to be the result of a disorder involving the mesentery. The upper part of the mesentery is especially likely to be involved. The process appears to be initiated by some trivial surgery. The intestinal obstruction which is found to be functional in nature occurs at some distance from the point of previous surgery and is noted most frequently after lower abdominal surgery. Surgery involving the upper abdomen is less likely to precipitate this process. Bonney noted that the functional obstruction bore no relationship to the magnitude of the inciting surgical process since it has occurred after simple clean procedures and had failed to appear following operative procedures of great magnitude. Sepsis seemed to have little or no causal relationship to it. Bonney believed that the pathologic changes associated with functional intestinal obstruction of the paralytic ileus type could only be explained on the basis of some agent or factor generated in the area of initial trauma conveyed to the neurovascular mechanism in or behind the mesentery either by the blood or lymph stream or along the nerve sheaths. He suggested that the factor was a product of tissue breakdown particularly where the blood supply to the tissues had been interfered with but not entirely cut off. This functional obstruction was also reported by Ponney as occurring in pregnancy. He reported operating upon patients who presented the clinical picture described for functional intestinal obstruction in whom no other cause could be found. He noted that his hypothesis of an agent producing functional intestinal obstruction has been strengthened.

ened by the publication of recent articles describing a functional or paralytic obstruction due to the administration of hexamethonium given to reduce blood pressure.

In recent years increasing numbers of reports have appeared describing a type of intestinal obstruction caused by the use of methonium compounds. Hexamethonium has been reported as causing a functional type of intestinal obstruction resulting in death. Pentamethonium bromide also has been reported as producing a functional intestinal obstruction. In a case of this type 100 mg of pentamethonium was given three times a day. At operation a picture of intestinal obstruction was presented in which the entire small bowel was distended as far as the ileocecal valve. The small bowel was found to be pale as though it contained less blood than usual. The ileocecal valve appeared to be in spasm. In the patients operated upon by Bonney the mesenteric vessels were noted to be engorged and the segment of bowel they supplied was purplish red in color, greatly distended with gas, and its upper portion heavily laden with fluid.

Pentamethonium bromide produces medical sympathectomy. In addition to blocking the sym-

pathetic ganglia, it also blocks the parasympathetic ganglia. Functional intestinal obstruction may be caused by this mechanism. The methonium salts are probably not metabolized and are excreted unchanged by the kidney. With renal impairment there is danger of accumulation of these methonium salts which would further restrict the urinary output. This vicious cycle becomes a lethal mechanism. The treatment of cases of this type consists of the injection of epinephrine or arterenol.

Chronic or Partial Small Bowel Obstruction

Chronic intestinal obstruction is that type of interference with the downward passage of intestinal contents of a sufficiently high grade to produce symptoms without entirely obstructing the bowel. The symptoms are due to the compensatory hyperperistalsis and hypertrophy of the musculature of the proximal bowel which attempts to overcome the blockage of the intestinal stream. This hypertrophy and proximal bowel dilatation in turn increase the extent and force of peristalsis.

Although surgeons are alert to the possible diagnosis of acute intestinal obstruction, an accurate diagnosis of incomplete (chronic) intestinal obstruction is infrequently made. This is due to the

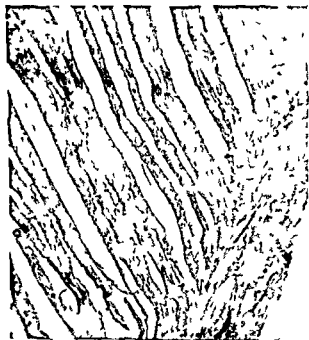


FIG. 129 Note the hypertrophy of the circular layer of the bowel as a result of a prolonged high grade intestinal obstruction.

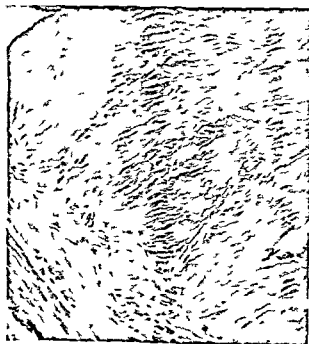


FIG. 130 Note the marked hypertrophy of the circular muscle layer with long standing intestinal obstruction.



FIG 131 Chronically obstructed bowel laid side by side. Note that the luminal diameter of the upper bowel is approximately $\frac{1}{4}$ the diameter of the lower bowel being held by the surgeon's hand.

indefiniteness of the symptoms and their variability. This type of obstruction usually escapes radiologic diagnosis.

The onset of chronic or partial intestinal obstruction is slow and insidious. The symptoms are disturbances of intestinal function repeated at tacks of obstruction and vague and bizarre abdominal complaints. The onset may be abrupt



FIG 132 Partial obstruction of the small bowel due to adhesions. Notice the pooling of barium.



FIG 133 High grade partial mechanical small bowel obstruction. Notice marked small bowel distention.

but the symptoms usually disappear rapidly. Colicky pain characterizing all interruptions to the intestinal stream comes on suddenly and usually arises at some fixed point. It radiates as a result of hyperperistaltic activity above the point of stenosis or partial obstruction. There may be an arrest of intestinal gases and also intestinal contents. Slight distention may or may not appear and the patient may vomit during this stage. The attack of obstruction is of brief duration. It may last from 24 to 48 hours and then all signs and symptoms disappear with a large evacuation. These episodes are repeated at varying intervals but tend to become more severe with the passage of time. Abdominal distention becomes more pronounced with the passage of time and the patient may complain of a sense of fullness. Between attacks of obstruction the patients characteristically are constipated with intermittent spells of diarrhea. Many lose their desire for food. Despite this history of loss of desire to eat all those patients personally observed by the authors appeared to be fairly well nourished—so much so in fact that the question of a psychosomatic overlay entered the picture. It may be very difficult to differentiate between a patient suffering from chronic intestinal



FIG. 134 The same patient as shown in Figure 143. Barium injected through the Cantor tube present far down the gastro-intestinal tract pools as it fails to pass into the colon. At operation several points of high grade partial obstruction were found and corrected.

obstruction and a psychoneurotic with gastro-intestinal fixation. For this reason psychiatric consultation is desirable in all such cases.

Radiologic study performed in the usual manner is of little or no value. The barium meal invariably passes through the bowel without disclosing any points of obstruction or dilatation of the small bowel lumen. The Schatzki technic in which the long intestinal decompression tube is passed into the proximal bowel and then the small bowel filled with barium suspension is an important advance in the diagnosis of such partial obstruction. Points of narrowing and areas of dilatation of the bowel may be brought out by this technic. The Abbott technic of measuring the intraluminal pressures by means of a Miller Abbott tube is also suggested as a possible aid in diagnosis. The pressure in the bowel proximal to such points of partial obstruction should be increased. A correct

diagnosis may be reached in most cases by correlating the clinical observations of the patient over a long period of time with the physical and laboratory findings.

Strictures of the Small Bowel Causing Chronic Obstruction. Small bowel strictures may result from a previous enterostomy. After removal of the enterostomy tube contraction may occur to such an extent that a high grade partial obstruction results. A classic case report of this type is the following:

M.D., age 51 white female was admitted to Grace Hospital on November 1, 1932 with a diagnosis of partial intestinal obstruction. She gave a history of having been operated upon for bowel obstruction in 1927. At that time lysis of adhesions was the operative procedure. In 1947 she suffered a recurrent episode of partial obstruction. She was reoperated upon and a bowel resection performed. At this time an enterostomy was performed proximal to the point of anastomosis. Since this last operation, the patient complained of intermittent inter-



FIG. 135 High grade partial small bowel obstruction due to adhesions from chronic adhesive peritonitis. This was the result of a diverticulum of the small intestine with ulceration of the mucosa and perforation at some previous time.

tinal distention. This became increasingly worse and was associated with cramping pain in the abdomen. She became progressively more constipated. On examination her abdomen was found to be markedly distended. The distention was found chiefly below the umbilicus and followed the transverse colon. Definite tenderness could be elicited over the lower right quadrant. A mass could not be made out. Upon opening the abdomen a tremendous number of adhesions involving the small bowel and omentum were found. The terminal ileum was matted upon itself. It was also matted to the middle of the transverse colon which was redundant and hung below the navel. On severing the adhesions from the ileum and from the transverse colon it was seen that the ileum involved in the matted area of adhesions presented a small contracted stenotic area barely large enough to allow the entrance of the index finger. The intestine was twisted upon itself and its serosal surface was densely adherent to loops of bowel adjacent to it. The bowel was markedly kinked and its wall was thickened and roughened by fibrous tags and fibrous adhesions.



FIG. 136 Same specimen as in Figure 135. Note the matting, kinking and angulation of the bowel. The arrow points to the site of diverticulum.

Among the rare causes of stricture and stenosis of the jejunum is the ingestion of ammonia. Lesions of the small bowel following the ingestion of either acid or alkali are rare. Only two cases of jejunal lesions due to alkali poisoning have been reported. Both cases were fatal. Chavassin and Shitkovsky reported a case of this type in which marked stenosis involving the upper jejunum produced a high grade obstruction with the result that the patient developed alkalosis and hypopotassemia. The constriction of the jejunum was 12 cm. long and the lumen of the bowel was shlike in character. The wall was three times its normal thickness in section. It was white and very firm. The mucosal surface presented a serpiginous ulceration and was covered by a white slough.

A not uncommon cause of stenosis of the small bowel is the type that occurs following X-ray therapy and radium therapy for carcinoma of the cervix. A post irradiation fibrosis may occur in



FIG. 137 Post irradiation fibrosis of the small bowel with stenosis as a result of X-ray therapy for carcinoma of the cervix.



FIG 138 Paralytic ileus as a result of perforation of a nonspecific ileal ulcer. There was walling off of this perforation with mechanical obstruction of the terminal ileum. Note the marked small bowel distention with the presence of gas in the colon indicating paralytic ileus.

the bowel causing high grade partial obstruction.

Nonspecific Ulcer of the Small Bowel

Among the rare lesions of the small bowel producing chronic intestinal obstruction is the nonspecific ulcer. This type of ulcer was first described in 1805 by Baillie. Little is known of this intestinal lesion. The clinical picture may be vague and confusing. As a result the preoperative diagnosis is almost impossible until a cicatrizing stenosing lesion appears due to healing of the ulcer causing partial intestinal obstruction. In some instances this ulcer perforates. A diffuse peritonitis, localized peritonitis or walled-off abscess results and a paralytic ileus follows. In some instances the perforation occurs so slowly that it becomes sealed off by adjacent loops of bowel. A matting together of the loops of bowel in the circumjacent area

then occurs. A complete or partial intestinal obstruction may be produced in this fashion.

In the management of lesions of this type resection of the matted bowel and end-to-end anastomosis of normal bowel are the procedures advocated. Those cases complicated by abdominal abscess or peritonitis are treated conservatively. In cases in which the adhesions involve a bowel length of such size that resection is contraindicated the Noble plication procedure may be of value.

Chronic obstruction of the terminal ileum or ilocecal region is not uncommon as a result of hyperplastic tuberculosis. This variety of tuberculosis was first described by Hartmann and Pillet in 1899. The degree of stenosis is usually not sufficient to produce complete obstruction. In an occasional case a mass of fibrous intestinal material upon reaching the narrowed tuberculous granulation tissue may create a ball valve type of



FIG 139 Obstruction of the terminal ileum produced by the pressure of a granuloma of undetermined origin. Note the marked narrowing of the terminal ileum.

complete intestinal obstruction. When this occurs, the contents of the terminal ileum which are usually liquid trickle through the small stenosed area for a long period of time. Fibrous foods or foods containing seeds may act as a nidus for the formation of a ball valve fecal bolus at the point of stenosis.

Chronic granulomas of unknown etiology may occasionally be found at the terminal ileum. These may cause chronic or partial intestinal obstruction. Invagination of the loop of ileum involved results in the production of an ileocecal intussusception.

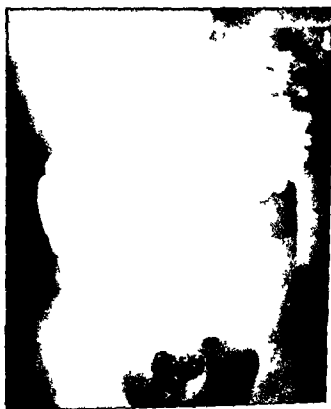


FIG 140 Same patient as in Figure 139. In addition to the compression of the terminal ileum with granuloma formation there was some intussusception of the terminal ileum into the cecum. Note the indentation in the cecum indicating the intussusception at this point.

OBSTRUCTION OF THE COLON

The surgical approach to obstructive lesions of the colon differs entirely from that used in small bowel obstructions. Colonic obstructions present few of the dramatic events that occur in small bowel obstruction. Unlike the small bowel the colon lends itself to diagnostic study. The sites of predilection of the obstructive processes of the colon are such that any physician can attain a high degree of accuracy in their diagnosis. A gloved index finger and the sigmoidoscope are sufficient for the diagnosis of 65 per cent of all colonic obstructions. No other portion of the body repays a carefully performed and systematic examination with such a high percentage of exact and accurate diagnoses.

There is some variation in the statistically reported incidence of obstruction with carcinoma of the colon. Incidences as low as 2 per cent and as high as 29 per cent have been reported. These rates depend to some extent upon the type of surgical patient treated and whether private or charity patients constitute the bulk of cases. It is obvious that acute colonic obstruction is not a common occurrence in malignant colonic disease.

With the exception of sigmoid volvulus the average duration of symptoms associated with obstructive colonic lesions may be as long as three to nine months. During this period of time the patient may show progressively increasing obstruction of an incomplete variety. When these lesions occur in the older age groups as they commonly do, obstipation is very difficult to evaluate since most of these oldsters consider constipation a normal feature of life. As a result considerable time may elapse before any medical attention is

sought. Moreover carcinoma of the colon may be present for a relatively long period of time before obstruction becomes complete and acute. This has been shown to be due in many instances to the marked edema and inflammatory reaction produced by the malignancy.

Acute obstructions of the colon may be broken down into three clinically recognizable groups:

- 1 In this group one finds all those patients in whom it is impossible to be certain whether the obstruction is small bowel or colonic or a combination of both.

- 2 In this group the patients are definitely known to have colonic obstruction but the point of obstruction and its etiology are unknown.

- 3 In this group not only is the colonic obstruction proved but the site of the obstruction and the nature of the obstructing mechanism are known.

In considering the diagnosis and management of obstructions of the colon the right and left colons must be considered separately since the manifestations of obstruction differ considerably in these two portions of the bowel. Because the right colon derives from midgut its physiology is much like that of the small bowel. The right colon is highly absorptive and its content is semi-solid. Its caliber is very large and its wall thinner than other portions of the gastro intestinal tract particularly the left colon. The bacterial content of the right colon is considerably lower than that found in the left colon. Right colonic lesions are often ulcerative growths of a polypoid nature. These often project far into the lumen of the bowel. Consequently unless the ileocecal valve is involved intestinal obstruction occurs relatively



FIG 141 Marked small and large bowel distention due to a carcinoma of the sigmoid with an incompetent ileocecal valve



FIG 142 Same patient as in Figure 141. Lateral view showing marked distention

late. Because of the ulceration of the polypoid growth aneurysm may be a very prominent feature. Infection caused by this ulceration is not uncommon in right colonic malignancies. Pericolonic infection may occur with the development of an abscess which may be indistinguishable from an appendicular abscess or a ruptured cecal diverticulum. In this event paralytic ileus may result.

The obstructive lesions of the right colon are usually more acute than those of the left colon and there is more small intestinal distention. The colicky pain and vomiting resemble those seen in small bowel obstructions. Moreover, although the long intestinal decompression tube can be successfully used to decompress the proximal bowel in obstructions of the right colon so that a primary resection and anastomosis can be performed as a one stage procedure, the long tube must never be used to decompress the left colon.

Obstructions are far less frequent in the transverse colon than in the right or left colon. Although the same etiologic factors are responsible for such obstructions, these factors are much less likely to obstruct the transverse colon both because of its anatomy and because of the decreased incidence of malignant lesions there when compared with other portions of the colon. The fixation at the hepatic and splenic flexures is also responsible for the negligible incidence of volvulus.

Obstructions of the left colon occur approximately nine times as often as obstructions of the right colon. Such obstructions may be acute or chronic, complete or incomplete. The narrow lumen of the left colon and its solid content with a high bacterial count are mainly responsible for the high incidence of obstruction in this portion of the colon. Left colonic obstruction, particularly those of the rectosigmoid, are characterized by tremendous distention of the colon. This is especially true of those cases where the obstruction is



FIG. 143 This is a good example of the use of the long intestinal tube in the management of obstructions of the right colon. Notice the presence of the tube head in the cecum.

been on a long standing chronic basis. Although interference with the mesenteric circulation is not a common feature of left colon obstruction, obstructions due to volvulus, intussusception or invasion of the inferior mesenteric artery or one of its main branches by neoplastic cells are exceptions to this generalization. Hypertrophy of the muscularis is usually a prominent feature of left colonic obstruction particularly when the acute obstruction has developed on a long standing chronic obstructive process.

The colon is subject to tremendous distention as a result of left colonic obstruction. Although this distention can cause capillary circulatory impairment resulting in cecal blowout, the colon itself is relatively immune to strangulating obstructions for the following reasons:

1. Obstructive lesions of the left colon are chiefly on a neoplastic or inflammatory basis arising within the bowel lumen. In such cases the

mesentery and its blood vessels remain unaffected by the obstructive process.

2. The only portion of the colon that ordinarily has a mobile mesentery is the sigmoid colon. The vessels of the remainder of the left colon are protected because the descending colon has no free mesentery and the colon is well fixed. As a result the left mesocolonic vessels are not likely to become obstructed. The sigmoid colon, however, having a long mesentery with a narrow base may undergo strangulating obstruction as a result of interference with the circulation. Volvulus producing a torsion of the mesosigmoid is a common cause of strangulating colonic obstruction.

DIAGNOSIS

Many patients with colonic obstruction give a history of intermittent constipation and diarrhea. In some a history of diarrhea, bloody stool, fever and mucus in stools may lead the unwary into a snap diagnosis of colitis. Although colitis and pericolitis may at times be present, the underlying primary lesion is often a carcinoma. Malignancy of the colon is associated with inflammation and colon spasm in a relatively high percentage of the acutely obstructed cases. Generally these patients report an increase in the number of stools per day. As many as three to seven small loose stools may be evacuated daily. This may be associated with rectal irritability and pain in the anal area or tenesmus. Gross blood, either alone or with mucus, appears in about half the cases. In a smaller number of cases, bloody stools may be the first symptom noted. Although a loss of weight is common, vomiting or nausea are very uncommon. In many cases a history of increasing girth around the waist is reported. Anemia and weakness may be associated findings. Often the patient's only complaint until the acute obstruction sets in is an increasing constipation of several months' duration.

The first diagnostic procedure after a carefully taken history and physical examination suggest the possible presence of intestinal obstruction is a survey film of the abdomen. This should then be followed by a definitely planned routine. Only in this way will errors in diagnosis be avoided. There are many who rely upon statistically gained knowledge to arrive at a presumptive diagnosis of the

etiology of the disorder. Although such a method is the equivalent of a snap diagnosis, it is none the less worthwhile to bear in mind the following statistics:

1 The commonest cause of obstruction of the colon in the middle aged group is cancer.

2 Malignancies account for approximately 90 per cent of all colonic obstructions, with diverticulitis second and volvulus third.

3 The commonest sites for obstruction of the colon are the sigmoid, rectosigmoid and rectum. These three sites account for about three fourths of all cases of colonic obstruction. The cecum comes next as a site of obstruction followed by the splenic flexure.

4 Diverticulitis when a cause of intestinal obstruction is almost invariably found in the pelvic colon.

Combining all this statistical data one may speculate that if a survey film of the abdomen indicates a colonic obstruction in a middle aged or old patient the probabilities are nine to one that the lesion is a carcinoma of the rectosigmoid, rectum or pelvic colon with a diagnosis of diverticulitis as a second choice. Although such statistically accumulated data are useful they must never be used as a short cut in making a diagnosis. Nothing short of a thoroughly performed physical examination plus a radiologic examination suffices in any case. We have found the following routine of examination most helpful:

1 Carefully taken history, noting particularly changes in bowel habits and changes in caliber or character of stool. Presence of blood or mucus in stool is especially to be questioned.

2 A carefully performed physical examination. This includes a rectal (and vaginal examination in females) and a sigmoidoscopic examination.

3 Survey film of the abdomen in the erect supine and lateral decubitus positions.

4 In those cases in which the obstructing lesion is proximal to the sigmoidoscopic range (25 cm) then a barium enema X ray is the next step in the diagnostic procedure. Great care must be exercised in the performance of such studies. If too much pressure is used in injecting the barium the contrast medium may be forced proximal to an incomplete colonic obstruction so that the retained

barium may then render the incomplete obstruction complete. For this reason the radiologist should be informed of the possibility of colonic obstruction. If diverticulitis is suspected as the obstructing mechanism barium enema studies should be deferred until the acute process subsides. This is especially important when an impending or actual perforation of a diverticulum or ulcerative carcinoma is a possible diagnosis. In such cases barium enema is contraindicated until all signs of the acute process have subsided.

By using the routine as outlined it is possible to correctly diagnose more than 80 per cent of all colonic obstructions. However, there is a small group of cases in which the barium enema demonstrates an obstruction proximal to the sigmoidoscopic range yet the exact cause of the obstruction cannot be radiologically determined. These lesions account for less than one tenth of all colonic obstructions. In this group one finds (1) cecal volvulus (2) obstructions of the colon due to adhesive bands (3) diverticulitis (4) submucosal tumors and foreign bodies in the colon.

In any case of obstruction of the colon it must be remembered that multiple carcinomas may be present. Thus an obstructing carcinoma of the hepatic flexure may be associated with a non-obstructing carcinoma of the rectum. In one instance an obstruction of the pelvic colon due to a previously performed Mikulicz operation was found to be associated with a high grade partial



FIG. 144 Carcinoma of the rectosigmoid and the ascending colon. This is an example of multiple primary cancers.



FIG 145 Carcinoma of the rectum producing no obstruction. Obstruction of the descending colon as a result of a previous Mikulicz resection and carcinoma of the hepatic flexure producing no obstruction. This survey film which shows marked intestinal distention, was made with a bedside unit and the quality of the film is therefore poor.



FIG 146 Same patient as in Figure 145. Note the point of obstruction in the sigmoid colon at the site of a previous Mikulicz. The carcinoma at the rectosigmoid can not be seen but is readily noted by sigmoidoscope. Note the large cauliflower carcinomatous mass at the hepatic flexure. There are three pathologic entities in this specific case.

carcinomatous obstruction of the hepatic flexure and a non-obstructing carcinoma of the rectum. Lesions of the rectum and the rectosigmoid are difficult to diagnose by X ray. They do however lend themselves well to sigmoidoscopic study. Considerable embarrassment to the surgeon and needless suffering for the patient may be avoided by following the survey film with preliminary sigmoidoscopic study in any case of intestinal obstruction. Even small bowel obstructions may be associated with colonic malignancies or may even be caused by such malignancies.

Laboratory studies are of very little help diagnostically except for the finding of anemia and the not infrequent elevation in the blood nitrogen.

ROLE OF THE ILEOCECAL VALVE

Any consideration of obstructive lesions of the colon cannot properly be made without taking

into account the physiology of the small bowel and the ileocecal sphincter. Distention of the small bowel as noted radiographically has been found to be present in approximately 30 to 40 per cent of all colonic obstructions. It is far more likely to be found in association with obstructive lesions of the right colon particularly those involving the ileocecal valve than with obstructions of the left colon. In this event the competence or incompetence of the ileocecal valve is of the greatest importance in evaluating the radiologic findings as well as in determining the type of treatment to be given.

There appears to be considerable diversity of opinion with regard to the incidence of competence of the ileocecal valve. Dennis found that this valve was competent in 61 per cent of a series of 54 colonic obstructions. The significance of such a high incidence of competent ileocecal valves lies

in the fact that this valve when competent is able to create a closed loop obstruction whenever obstruction distal to the cecum occurs. This creates a definite danger of cecal blowout. However, since cecal blowout occurred in only one patient in a series of 126 consecutive colonic obstructions treated at Grace Hospital we believe that a re-evaluation of the question of ileocecal sphincteric competence is indicated.

The anatomy of the ileocecal valve is such that distention of the cecum by stretching of the lips or folds above and below the valve tends to increase its competence. In addition it has been demonstrated that the ileocecal valve is subject to definite nervous control. Its competence appears to depend to some extent upon the tonicity of the fibers of the sphincter. The tonus of this sphincter has been increased experimentally by stimulation of the sympathetic nerves. In addition stimulation of the distal part of the colon increased the back pressure to approximately three times that of the normal sphincter. Stimulation of the parietal peritoneum, stomach and small bowel had no such effect. These experimental studies suggest that the resistance of the ileocecal sphincter to back pressure is greatly increased in cases of intraluminal pathologic conditions of the distal colon. Stimulation of this portion of the colon acting through Auerbach's plexus is believed to increase the tonus of the ileocecal sphincter making it more competent. These observations would all seem to lend further support to the danger of closed loop obstruction and cecal blowout in association with a competent valve.

However anatomic studies demonstrate that although the normal adult cecum presents a competent ileocecal valve there are varying degrees of valve competence associated with variations in the cecum. The cecum is slow to assume its adult form in humans. As a result large numbers of imperfectly developed ceca are found; there is of necessity a concomitant variation in the degree of competence of the ileocecal valve. Contrary to popular belief perfectly functioning competent valves are in the minority. Reviewing the competence of this valve using a reflux of barium into the ileum during the performance of barium enema without undue pressure, radiologists reported that

65 to 90 per cent of the valves were incompetent. This would suggest that the majority of ceca are not perfectly developed in man. From the studies it would seem that closed loop obstruction can be possible only in this minority group (10 to 35 per cent of all cases) which has been shown radiologically to present incompetent ileocecal valves. Although the range between 10 and 35 per cent appears to be wide it can be explained by the fact that this review was the composite work of many radiologists and as is true in all such surveys the wide variation in personal abilities and capabilities must be taken into account. However the most highly trained radiologists were almost unanimous in reporting 90 per cent of the ileocecal valves incompetent.

In 1935 Saeltzer and Rhodes reviewed the literature available on perforation of the cecum. They defined this condition as a blowout of the wall of the cecum produced by overdistention caused by obstruction of the colon distal to it. The term diastatic perforation of the cecum was first used by Heschl in 1880. From this we must infer that this lesion has been known for many years. Despite this fact fewer than 100 cases were found in the literature by Saeltzer and Rhodes. Rack reviewed 25,020 admissions to the surgical service of the Cleveland City Hospital. In this group 509 patients were admitted with carcinoma of the colon. Of these perforation of the cecum had occurred in 1.5 per cent. This figure tallies with our experience at Grace Hospital. It is possible that many similar cases have occurred but have not been reported. Although the relatively few reported cases may not reflect the true incidence of this complication our experience leads us to believe that diastatic perforation of the cecum is a relatively uncommon accident. Such perforation of the cecum presupposes not only obstruction of the colon distal to the ileocecal valve but also a competent ileocecal sphincter which prevents the backflow of intestinal colonic contents into the terminal ileum. Diagnostically in the presence of a perforation of the cecum secondary to left colonic obstruction the survey film of the abdomen may reveal large amounts of subphrenic air. In an occasional case no air is found below the diaphragm. The colon may or may not

be markedly dilated depending upon the size of the perforation in the cecum. The perforation of the cecum is usually small and may even be pin point. In an occasional case multiple small perforations of the cecum may be found at operation. The presence of a pneumoperitoneum with air under the diaphragm is indicative of a perforated viscus. When a tremendously dilated and distended cecum and colon are added to this the radiologic appearance is pathognomonic of perforation of the cecum. The perforations are usually found on the anteromedial aspect of the cecum although at times any portion of the cecum may perforate. Widespread peritonitis is the rule in such cases so that peritonitis may be obvious on physical examination. The radiologic evidence of small bowel distention found in such cases would be the result of paralytic ileus. A similar radiologic appearance may occur in an occasional case where cecal perforation has not occurred but in which the ileocecal sphincter is competent and the obstruction is of long duration. As an additional confusing factor the small bowel may actually be mechanically obstructed as a result of the adherence of a loop



FIG 147 Note the pneumoperitoneum as shown by the accumulation of air under the diaphragmatic leaves



FIG 148 The long intestinal decompression tube has passed through the ileocecal valve and is now seen in the sigmoid colon

of small bowel to the malignant process which has invaded the colonic serosa. A similar type of obstruction with a similar radiologic appearance occurs when a loop of small bowel becomes adherent to a metastatic lymph node in the mesentery. This type of combined large and small bowel obstruction is not uncommon in obstructions of the sigmoid colon due to advanced carcinoma.

ETIOLOGY

The colon can be obstructed in many ways by varied etiologic factors. Although the same factor may produce obstruction in any portion of the colon there is a wide variation in the incidence with which it occurs.

Adhesions

Congenital or acquired adhesions may occasionally cause obstruction of the right colon. The



FIG 149 Carcinoma of the sigmoid colon producing intestinal obstruction. Note the position of the intestinal tube head in the ascending colon.



FIG 150 Carcinoma of the rectosigmoid with intestinal obstruction. Note the preliminary use of the long intestinal decompression tube rendering primary resection and anastomosis much simpler.

most common point of obstruction is the hepatic flexure. Acquired adhesions may occur postoperatively or may be the result of infection in the right upper quadrant secondary to lesions of the gall bladder or stomach. Pericholecystic abscesses from the perforation of the gallbladder may be followed by colonic obstruction due to adhesions. Subphrenic abscesses secondary to perforated ulcers or ruptured appendixes may produce such obstructing adhesions as late sequelae. It has been shown experimentally and substantiated clinically that strictures may result from extensive trauma to the abdominal wall. If injury to the bowel predominates then the ensuing strictures may be caused by submucosal or subserosal hemorrhage. If the injury occurs in the mesentery a mesenteric hematoma may dissect into the wall of the bowel and cause fibrosis or so injure the vessels that the intestine suffers from a circulatory lack which in turn causes fibrosis. Schloffer reported one case

in which complete obstruction occurred several years after trauma. He believed that a true stricture of the intestine following trauma occurred only in the small bowel. Burnham however reported a case of obstruction of the upper ascending colon caused by a trauma to the ascending colon which had occurred 40 years previously. At operation the ascending colon was found to be obstructed by dense fibrous bands which compressed it. These bands were vascular and had to be freed from the bowel by sharp dissection. In a review of the literature up to 1930 207 references to abdominal trauma producing strictures resulting in intestinal obstruction were found. It would therefore be wise in any case of intestinal obstruction with no evidence of previous surgery to include a question as to whether trauma had been applied to the abdomen at any time in the patient's life.

Although obstruction of the transverse colon by adhesions is uncommon, both congenital and acquired adhesions may on occasion produce partial obstruction of this portion of the colon. Draper and Johnson noted that in some instances developmental anomalies occur as a result of the migration of the cecum coupled with the simultaneous delamination of the omental layers. This can produce an abnormal implantation of the omentum. Dense and fibrous contracture bands can then occur which may cross or surround certain segments of the colon. In this fashion intestinal obstruction of the hepatic flexure, the ascending colon or the hepatic portion of the transverse colon may be produced. High grade partial obstruction of the transverse colon may occasionally occur as a result of adhesions between the greater omentum and the pelvic structures after pelvic surgery. We have observed instances at operation in which the transverse colon was sharply angulated by this mechanism. The basis for this type of partial obstruction is set at the time of pelvic surgery by the current practice of tucking the greater omentum behind the pelvic structures. From our experience we believe that this practice should be discontinued. Even when angulation of the transverse colon is not produced, the bridge formed by the adherent omentum to the pelvic structures furnishes an axis about which small bowel looping and obstruction may occur.

The transverse colon may be compressed against the posterior abdominal wall by the proximal limb in a gastrojejunostomy where the technic used involves suture of the proximal limb to the lesser curvature and the distal limb to the greater curvature anticolically. Any anticolice anastomosis may cause colonic (transverse) compression but the variety referred to is more likely to do so.

Adhesions may produce obstruction of the left colon. Among the more unusual causes of complete obstruction of the sigmoid colon are congenital fibrous bands. These may spring from the right of the descending colon and sweep across it attaching to the psoas muscle on the left. In one case of this type reported by Mainzer, the degree of colonic distention was so great that it involved the appendix. Gangrenous changes resulting from the appendiceal distention occurred at the tip of

the appendix which perforated and caused a fatal peritonitis. Traumatically produced adhesions can cause left colonic obstruction. In some instances a traumatic hematoma may so compress the colon that obstruction is produced. In cases of hemorrhage into the mesosigmoid involving the sigmoid colon when organization of the clot occurs there may be extensive adhesions between the small bowel and the colon. This may result in obstruction of the small bowel, the colon, or both. Jones reported an unusual case in which trauma to the abdomen produced a massive hematoma in the mesentery of the left colon. This dissected its way to the colon. The inflammatory reaction fol-



FIG 151. Obstruction of the descending colon by compression of adhesions following hemorrhage.



FIG. 152 Same patient as in Figure 151. Obstruction of the colon due to nonpenetrating trauma with hemorrhage. This radiograph demonstrates the eccentrically situated stenosing lesion of the midportion of the descending colon producing obstruction.

Following the hematoma produced a compression of the colon obstructing it. An eccentrically placed defect was noted radiologically in the bowel wall. This coupled with the history of trauma, lack of blood in the stool, absence of weight loss, and the general appearance of the patient was considered sufficient evidence for an accurate diagnosis.

Foreign Bodies

Obstruction of the right colon as a result of ingested foreign bodies is rare. Generally such

ingested foreign bodies are excreted per rectum once the ileocecal valve has been successfully passed. However, this happy outcome does not always occur. As far back as 1893 a specimen of obstruction of the cecum caused by a foreign body was exhibited at the Pathological Museum of the British Medical Association. In this case an ingested dessert spoon 7 inches long, with a bowl $1\frac{1}{2}$ inches across had passed partly through the ileocecal valve and partially obstructed the cecum. The colonic mucosa had undergone considerable ulceration. Wingunsteen and Harris each reported obstruction at the ileocecal valve as a result of a portion of the balloon of the air-filled intestinal decompression tube becoming arrested in passage through the valve. In rare instances obstruction at the hepatic flexure of the colon may occur because of a gallstone. In such cases a cholecystocolonic fistula can permit gallstones of large size to erode through the colon at the hepatic flexure. Generally such stones are excreted per rectum.



FIG. 153 Cholecystocolonic fistula, treated by a barium enema.

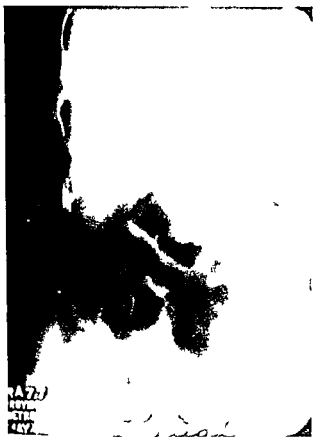


FIG 154 The same patient as shown in Figure 153. Note the gas filled ile radical which are indicative of cholecysto-enteric fistula.

In an occasional case arrest of stones occurs as a result of bowel spasm.

Foreign bodies rarely cause obstruction of the transverse colon. Almost invariably such intracolonic foreign bodies pass through the transverse colon without incident. If the transverse colon is angulated or compressed and thus partially obstructed then a foreign body may become arrested and cause obstruction at this point. In rare instances foreign bodies of very large size may become arrested in the transverse colon. An interesting example of this was reported by G. C. Turner. In this case the transverse colon was completely obstructed by two faceted gallstones. The larger stone weighed 4800 grains and measured 3 by 2 $\frac{1}{4}$ by 7 inches in circumference. The smaller stone weighed 960 grains and measured 1 $\frac{3}{4}$ by 1 $\frac{1}{2}$ by 4 $\frac{1}{2}$ inches. An analysis of these stones proved them to be gallstones composed of cholesterol and bilirubin pigments.



FIG 155 Cholecystocolic fistula. Note the barium filled biliary radicals.

Various types of foreign bodies introduced per rectum have been removed from the sigmoid colon because of the obstruction they produced. Among these are preserve bottles, pop bottles, stones, and wine glasses. Colotomy with removal of the foreign body has been required where the objects were so large that they could not be removed from below. In the sigmoid colon gallstones that are not unusually large may produce intestinal obstruction. The presence of the stone irritates the colonic mucosa causing a spasm in the sigmoid obstructing it. Among the foreign bodies causing obstruction of the left colon the most unusual is a case reported by Trevor in which the patient developed an obstruction of the descending colon as a result of swallowing air. As far as is known this is the only report of such a case. The patient had had a total laryngectomy for carcinoma of the larynx four years previously. In order to speak he used an esophageal voice. This necessitated the swallowing of large quantities of air, the sound of which when erupted could be converted into



FIG 150 Same patient as in Figure 150. This is a good example of cholecystocolic fistula. Again note the barium filled biliary radicals produced by the barium enema.

speech by the lips teeth tongue and palate. The mechanism of obstruction was due to the increasing distention of the proximal descending colon which caused the sigmoid colon to fold over on itself near its peritoneal attachment. This created a mechanical obstruction.

Infections

Amebic granulomas not uncommonly involve the cecum or ascending colon. A granulomatous tumefaction develops which may partially obstruct the bowel. The lumen of the bowel becomes markedly narrowed by the tremendous thickening of the colon wall due to the granulomatous process. Radiologic examination reveals a deformity or filling defect which involves a much longer area than would carcinoma. However, in some instances the defect may be localized and thus simulate carcinoma from which it may be differentiated only with considerable difficulty. A correct diagnosis is of great importance because the

treatment is entirely medical. Antiamebic drugs including emetine hydrochloride, Diiodoqu, fumagillin, chloroquine, oxytetracycline and chl tetracycline are most effective when used in combination. With proper medical management granulomatous lesions generally disappear within a month. Surgical treatment is reserved for complications such as intussusception, perforation, complete obstruction.

Tuberculosis commonly involves the ileocecal portion of the gastrointestinal tract. In the hypoplastic type of tuberculosis, a granulomatous mass involving the cecum or ascending colon may be found. The doughy consistency of this type of granuloma is said to distinguish it from neoplasms. Practically, such differentiation may be difficult without a corroboratory biopsy. The obstruction in such cases is usually incomplete. The treatment is entirely medical except when complications occur. Surgical intervention is then indicated.

Granulomas of undetermined origin may on rare occasions produce obstruction of the right colon. These obstructions are usually not complete. The tumefactions presented at operation are difficult to differentiate from malignant lesions. Morris described a granuloma at the hepatic flexure in which the granulomatous nature of the lesion was suggested by the extensive invasion of the mesentery, far out of proportion to that usually seen with any but the most advanced neoplastic disease. He noted in addition that the obstruction was caused by compression of the lumen by the granulomatous involvement of the colonic wall. The mucosa was intact. Buckstein noted that an intact mucosa observed radiologically in the presence of an obstructing lesion should be suspected of being a benign granuloma.

The transverse colon and left colon are singularly free from obstruction based upon infection, except for the infrequent amebic granulomas that may involve the sigmoid colon producing partial obstruction.

Neoplasms

Benign tumors are rarely reported in the right colon and reports of bowel obstruction by such tumors are extremely rare. Among these tumors one finds lipomas, fibrolipomas, fibromas, myxo-

mas and chondromas. Carcinoids are classified with the histologically benign tumors. This tumor however does metastasize in about 16 per cent of cases. When carcinoid tumors are in the ileum however the incidence of metastasis has been reported as ranging from 18 per cent to 82 per cent. Carcinoid tumor involving the ileocecal valve results in intestinal obstruction resembling small bowel obstruction. The obstruction differs in one important respect however in that its mode of onset is usually more acute. This is typical of all neoplastic involvement of this structure. The radiologic findings noted on the survey film may be indistinguishable from small bowel obstruction. Carcinoid of the cecum may reach a great size before obstruction occurs. In many cases the obstruction is precipitated by secondary inflammatory reaction in the bowel. In such cases the onset is slow and insidious. Distention and vomiting occur relatively late in the course of the obstructive process. The symptoms chiefly pain of a cramping character, hyperperistalsis, mild distention and a loss of desire to eat may develop over a period of three days. A common diagnostic error in these cases is appendicitis. This error is especially apt to be made in those patients seen late in the course of the disease when marked distention, vomiting, fever and leukocytosis are not uncommon. In the early days of the obstruction the temperature and blood studies are normal. Leiomyosarcoma, liposarcoma, ganglioneuroma or retroperitoneal tumors of any type may occasionally produce intestinal obstruction by so compressing the ascending colon as to stretch it markedly over on its anterior surface. The colonic lumen may become compromised as a result of this extrinsic pressure.

Benign tumors are rare causes of obstruction of the left colon. The pedunculated type of benign tumor may on occasion act as a nidus for the development of intussusception. The retroperitoneal tumors noted on the right side can on rare occasions obstruct the left colon when they are found in the left retroperitoneal area.

Malignant tumors obstruct the left colon nine times as often as they do the right colon. Usually the tumor found in the right colon is a fungating type which protrudes into the lumen of the bowel. Because the right colon is anatomically large in diameter with a thin wall obstructions due to

tumors are a relatively late development. Disturbances in intestinal physiology or bleeding are probably among the earliest signs. These patients often give a history of long standing chronic obstruction in which pain over the abdomen was felt for some time with increasing constipation and distention. Right colonic lesions produce obstruction as a result of four possible mechanisms: (1) the presence of the tumor mass, (2) a fibrosing or stenosing proliferative process which narrows the lumen, (3) inflammatory swelling and edema produced in the bowel by the neoplasm, (4) intussusception. That inflammatory swelling and edema play an important role in the production of right colonic obstructions can be demonstrated by the rapid relief of the obstruction when a diversionary procedure such as cecostomy or ileocolostomy is performed. As a result of the diversion of the fecal stream the inflammatory process often subsides so rapidly that a resumption of the normal fecal stream is possible.

In addition to primary carcinoma, metastatic



FIG. 157 Obstruction of the transverse colon secondary to metastatic carcinoma of the breast.

neoplasms may also produce right colonic obstruction. A case of this type was reported by Mahoney and Budd. In this patient a primary carcinoma of the pylorus metastasized to the terminal ileum obstructing the right colon and also metastasized to the rectosigmoid obstructing it.

There are three generally recognizable types of carcinoma involving the colon.

1 The fungating or ulcerative type most commonly is found in the right colon and cecum. This type has a tendency to ulcerate very early. It is a bulky tumor and is usually very friable and hence bleeds easily. Anemia is a prominent feature of this type of neoplastic involvement. In the right colon lesions of this type produce obstruction late.

2 The stenosing type of scirrhous carcinoma is not common on the right side although it is the most commonly found tumor in the left colon. This tumor tends to encircle the colon producing annular constriction. It has been estimated that



FIG 158 Intestinal obstruction caused by carcinoma of the right colon just below the hepatic flexure. Note the marked small bowel distention which may mask the colonic obstruction.



FIG 159 Barium enema given to the patient demonstrating obstruction at the hepatic flexure.

16 months may be required before complete encirclement of the colon occurs.

3 Carcinoma colloidum is an adenocarcinoma characterized by the production of a mucoid discharge.

In addition to these single recognizable types of carcinoma it must be remembered that multiple primary carcinomas may be found in the colon. For this reason whenever one colonic carcinoma is found a search should be made for other possible foci.

Lymphosarcomas are uncommon in the colon. When they are present the cecum is the most common site of growth. Since the new growth in the cecum is usually polypoid in appearance it may be recognized radiologically. These lesions tend to remain confined to the bowel for an appreciable period of time and metastasize relatively late compared to carcinoma. For this reason early operation is definitely indicated particularly when the ileocecal valve is involved by the neoplastic process. In such cases acute intestinal obstruction makes its appearance relatively early. In the surgi-

cal treatment of lesion of this type it has been shown that the removal of the primary tumor and whenever possible the involved lymph nodes should be the procedure followed. Although there is some difference of opinion as to the effectiveness of radiation therapy the consensus appears to advise radical colectomy followed by irradiation in all such cases. Cecal lesions of this type are reported as having the highest 5 year survival rate although in general the results are disappointing. Nitrogen mustard has been found to be of some value in the treatment of recurrent lymphoma which has become resistant to further X-ray therapy.

The prognosis for carcinoma of the right colon when diagnosed is usually better than for carcinoma of the left colon. This may be due to the fact that in excision of this tumor right hemicolectomy including the terminal ileum is the

procedure commonly used. With this technique the entire right colon and all its lymphatics are removed. This is a more radical procedure than that usually performed on the left colon.

Neoplasms of the transverse colon are generally partially constricting and present themselves as a well defined mass which is often readily palpable. It is not uncommon for the small intestine to adhere to the obstructive lesions of the transverse colon particularly when the serosa of the colon has become involved. In such cases a large bowel and small bowel obstruction may occur simultaneously. It is of the greatest importance to recognize this occurrence. Cecostomy to decompress obstructive lesions of the transverse colon would be quite useless when the small and large bowel are simultaneously obstructed. Obstructive malignancy of the splenic flexure is usually seen late in the course of the disease. The symptoms before the onset of the obstruction are usually vague and abdominal in character. Since the lesion is high under the left costal arch it is not palpable. Because of this acute obstruction may be one of the earliest symptoms of this disease. In some cases a barium enema may be misleading because the proximal limb of the splenic flexure may lie anterior to the distal limb and thus show no deformity in non-obstructive cases. For this reason whenever a neoplasm is suspected in this area an oblique exposure should be made so that the splenic flexure may be well visualized and the two superimposed limbs may be separated.

Carcinoma has been found to be responsible for approximately 80 per cent of all obstructions of the left colon. Not all carcinomas in this area are obstructive however in fact only 10 per cent of all carcinomas of the colon are found to cause obstruction. Blockage of the lumen of the left colon by carcinoma is brought about by various mechanisms. The following have been recognized:

- 1 The presence of a polypoid neoplastic mass within the colon may obstruct it or may cause intussusception.
- 2 Inflammatory swelling and edema may be superimposed upon a neoplastic colonic process converting a partial obstruction into a complete one.

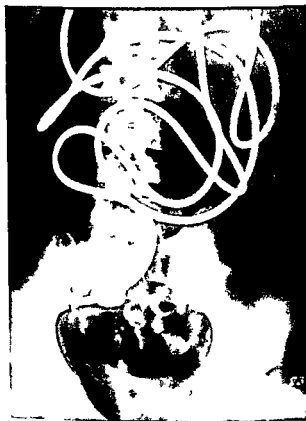


FIG 160 The use of a long intestinal decompression tube at the ileocecal valve to decompress the small bowel in obstructions at the ileocecal valve or low in the right colon.



FIG 161 Carcinoma of the descending colon. Small bowel distention treated with long intestinal decompression tube. The ileocecal valve is incompetent. Note how ever the distention of the right colon.



FIG 162 Lateral view showing tremendous distention associated with carcinoma of the rectosigmoid.

3 The carcinoma may encircle the bowel obstructing it in a ring fashion.

4 An incomplete colonic obstruction may become complete as a result of fecal impaction.

5 Metastatic tumors may obstruct the colon by direct extension from the peritoneum to the serosa of the bowel where compression is caused by the tumor mass.

6 The colon may be completely encircled by a carcinoma extending directly from the ovary or fallopian tube.

7 The sigmoid colon may become obstructed by invasion from a carcinoma of the cervix.

Obstructive lesions of the left colon are not uncommonly associated with acute inflammatory processes superimposed upon the obstructive neoplasm. Radiologic diagnosis in such cases may be erroneously interpreted as demonstrating inflammatory disease whereas the true underlying pathol-

ogy is carcinoma. The following case is a typical example.

A 57-year-old white female was admitted to Grace Hospital Feb. 9, 1949, with a diagnosis of intestinal obstruction. A barium enema given with considerable difficulty because of severe abdominal pain demonstrated a persistent narrowing in the sigmoid colon. A palpable mass corresponding to the sigmoid was noted. The radiologic findings were those of an intrinsic lesion in the sigmoid colon measuring approximately 16 cm. A diagnosis of inflammatory disease with sigmoiditis and stenosis of the colonic lumen was made. At surgery during the performance of a defunctionizing colostomy a medullary carcinoma was found in this area associated with secondary inflammatory changes.

As a result of the not infrequent inflammatory reaction fixation of the colon is commonly associated with obstruction due to carcinoma. Because of this a loop of small bowel may become adherent to the neoplastic process and as a result



FIG 163 Carcinoma of the rectosigmoid associated with a competent ileocecal valve. Notice the tremendous distention of the right colon. The long intestinal decompression tube should never be used in this type of case. Immediate defunctionizing colostomy is required in order to prevent cecal blowout.

small bowel obstruction occurs. The uterus, ovaries or Fallopian tubes may become adherent to the inflammatory obstructing carcinomatous process so that their removal is required along with the obstructing neoplasm.

Acute diverticulitis with or without perforation and abscess formation may occasionally be found associated with carcinoma of the colon. In such cases diagnosis and treatment tax to the utmost the diagnostic and therapeutic judgment of the attending surgeon. The paralytic ileus due to the infectious process may mask the radiologic findings of mechanical intestinal obstruction, especially if one depends upon the survey film alone.

The signs and symptoms of acute obstruction of the left colon due to neoplasms are essentially the same as those of acute obstructions low down in the gastro intestinal tract in the absence of strangulation. Except in the extremely infrequent



FIG 164 Carcinomatous rectosigmoid which produced obstruction.

cases of vascular occlusion by neoplastic invasion, strangulation of the left colon is rare with lesions of this type. Because the obstruction is low in the gastro intestinal tract, the symptoms are less fulminating and less acute than at other points in the colon. Considerable time may elapse from the time of onset of symptoms until the time when the patient is subjected to surgery. Such patients are often admitted to the hospital with a history of pain, distention and obstipation of 7 to 10 days duration. It is difficult in such cases to know at which point the obstruction became complete, but the majority probably were not completely obstructed during the entire 7 to 10 day period. The pain associated with the presence of an obstructing neoplasm is usually not severe, but it may be constant. This pain is often localized in



FIG. 165. Acute obstruction of the lower sigmoid due to carcinoma. The obstruction was of sudden onset and not heralded by any prior complaints.



FIG. 167. Carcinoma of the left colon with obstruction. There is little or no small bowel distention as yet.



FIG. 166. Carcinoma of the descending colon with obstruction. Note that the right colon is filled with feces and that there is little or no small bowel distention.

the region of the navel and is colicky in nature. Obstruction and distention are the most prominent symptoms.

The best type of surgical procedure to be used in the management of malignant obstructions of the colon depends to some extent upon the exact distance of the neoplasm from the anus. In many instances, an exact anatomic localization may not be possible until the abdomen is opened. The radiologic findings may at times be confused by the curve of the redundancy of the pelvic colon. It is not uncommon to get a radiologic report indicating a stenosing lesion in the sigmoid at 35 to 40 cm from the anus while sigmoidoscopic examination demonstrates the lesion to be 12 to 15 cm from the anus. Although the correlation of the radiologic and physical findings may be difficult, the importance of knowing just where the obstruction really is cannot be underestimated.

Volvulus

Volvulus of the cecum was first described by Kokotkin in 1841. Jackson reported a case ex-



FIG 168 Lateral view of obstructing carcinoma of the sigmoid colon



FIG 169 Note the marked small and large bowel distention in association with an obstructing carcinoma of the rectosigmoid

hibited to the Pathological Society in 180. In 1868 Lagge reviewed 4000 consecutive autopsies and reported four cases of volvulus of the cecum. Ziegler and Manteuffel reported 24 cases in 1898 and reviewed the literature up to that time. By 1913 304 cases of cecal volvulus had appeared in the literature. Fagel and Fagel reviewed the literature in 1923 and including 14 cases of their own found a total of 471 cases of cecal volvulus reported to date. The incidence of this form of obstruction appears to be a controversial subject, however, as indicated by the wide variation quoted for this condition by different authors. An incidence as low as 0.1 per cent and as high as 6 per cent of all obstructions has been ascribed to cecal volvulus. The true incidence probably lies between 1.5 and 3 per cent.

Catellier, Moutier, and Porcher define volvulus of the cecum as a condition in which the torsion

is limited to the cecum, ascending colon, and terminal ileum. This appears to be a satisfactory entity on embryologic and anatomic grounds because the right colon is usually fixed in its upper portion regardless of how defective the peritoneal attachment below the hepatic flexure may be. Volvulus of the cecum is possible only when certain conditions are present: (1) absence of fixation of the cecum; (2) a mesocecum sufficiently mobile to permit torsion; and (3) a fixed point about which the bowel can rotate. These derangements of the right colon are dependent upon incomplete fusion of the peritoneal layers of these parts. The cecum becomes fixed in the right lower quadrant as part of the final process of intestinal rotation. This occurs as a result of the more or less complete fusion of the peritoneal layers of the ascending colon and cecum. The movements of rotation, descent, and fusion of the ascending colon to the posterior abdominal wall may be inter-



FIG 170 Obstruction of the rectosigmoid associated with tremendous small and large bowel distention

rupted at any point. The most common anomaly due to derangements of this mechanism is a failure in fusion of the ascending colon which results in its having a mesentery. This increases the mobility of the right colon and renders it susceptible to torsion. The incidence with which such cecal mobility due to nonfixation occurs has been variously reported. Chalfont reported a 20 per cent incidence. Treves reported a 25 per cent incidence. G. M. Smith and S. C. Harvey reported an incidence of 32 per cent and 12 per cent respectively. In a comprehensive anatomic study, Wolfer, Beaton and Anson found that 11 per cent of all ceca were mobile enough to permit the development of a volvulus. In addition, a review of the literature indicated that 10 per cent of the cases presented a fixed terminal ileum about which volvulus could occur. The anatomic arrangement which best predisposed to cecal volvulus was considered to be one in which the cecum was free and mobile and the ascending colon was fixed somewhere along its

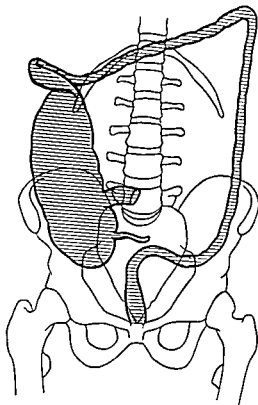


FIG 171 Volvulus of the cecum 360° torsion

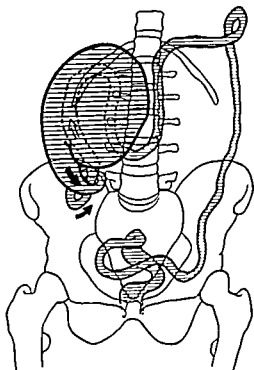


FIG 172 Volvulus of the cecum

vertical course the most common point being the hepatic flexure. The mechanism of volvulus varies widely. In general it appears to depend upon the anatomic relationship of a mobile cecum or colon associated with a fixed point at either the hepatic flexure or at the terminal ileum. The recognizable types of cecal volvulus are

1 When the volvulus occurs along an axis at right angles to the longitudinal diameter of the ascending colon so that the cecum and ascending colon are bent upon themselves

2 When the cecum is rotated in a clockwise or counter-clockwise direction on its long axis with the hepatic flexure as a central pivot

3 When the mesocolon of the ascending colon is unusually long and the right colon becomes twisted on its mesenteric axis

4 When a redundant loop of cecum with a long mesentery becomes intertwined among some loops of intestine and thus becomes obstructed

Volvulus of the cecum is essentially a disorder of young adults. It may however occur at any age. The youngest case was a 4-day-old infant reported by Borow and the oldest a 90 year-old patient reported by Manoil. This type of obstruc-

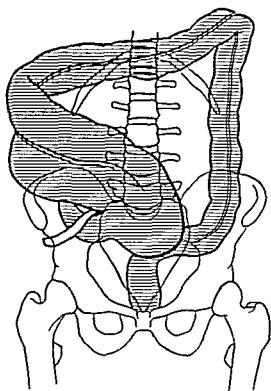


FIG. 174 Volvulus of the cecum 180° torsion

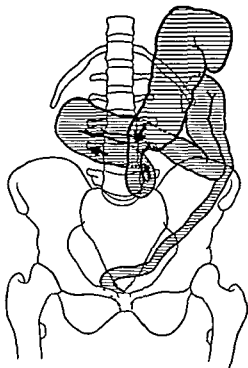


FIG. 173 Volvulus of the cecum

tion is reported to be more common in males than in females. However in the review by Figiel and Figiel the reverse was found to be true.

Among the precipitating factors responsible for the onset of cecal volvulus the following have been implicated: abdominal surgery with replacement of loops of bowel at the conclusion of operative procedure; pregnancy; violent exercise; a diet of heavy coarse foods; hyperperistalsis; intra-abdominal tumors; withdrawal of laparotomy packs; postoperative adhesions; and lithopedion.

The cecal volvulus may be acute or recurrent in type depending upon the degree of circulatory embarrassment and the arc through which the right colon swings as well as upon the tightness of the loop so developed. If the onset is acute the symptoms are those of acute mechanical bowel obstruction. Pain of a cramping character, vomiting, obstipation after the distal colon has been emptied and abdominal distention complete the picture. On physical examination a hugely dilated cecum which can be demonstrated radiologically on a survey film of the abdomen is readily noted. Kirby reported a case in which the

distended cecum was 45 cm long by 30 cm in diameter. At operation the greatly enlarged distended cecum may be found in any portion of the abdomen even in the left upper quadrant. There is often a history of previous episodes of generalized colicky abdominal pain, vomiting, obstipation, and rapid distention. The distention in such cases may reach tremendous proportions because cecal volvulus represents a closed loop type of obstruction. This distention cannot be relieved by vomiting, enemas or the use of a successfully passed long intestinal decompression tube. In the early stages of the obstruction violent peristaltic activity may occur so that borborygmi are quite audible. With the progression of the distention and impairment of the blood supply to the colon the signs of peritoneal irritation make their appearance. These are abdominal tenderness, rebound tenderness, rigidity and diffuse abdominal soreness. There may be an elevation of temperature and the patient may present a relative or an absolute leukocytosis. If the twist is very tight and the onset acute the symptomatology is apt to be more severe. This is particularly true when circulatory impairment is present. In such cases the patient may present some degree of shock. It is generally agreed that the torsion of the bowel upon its mesenteric attachment must be at least 180 degrees before pathologic changes begin. When pathologic changes do occur the venous circulation quickly becomes disturbed. A marked venous congestion occurs. This then causes hemorrhages into the tissues of the bowel and finally into the peritoneal cavity. If the process continues strangulation of the colon follows. In many instances of acute cecal volvulus the obstruction of the right colon is complete or nearly complete as a result of the torsion but there may not be impairment of the blood supply to the involved loop because of the looseness of the mesenteric twist. In the subacute type of volvulus the twisting is not of sufficient degree to produce a high grade obstruction. Since the degree of obstruction is not too great the symptoms of obstruction are not too marked. The twist in the bowel in such cases may relieve itself spontaneously only to recur at a later time. Figiel and Figiel demonstrated detorsion of volvulus of the cecum radiologically in three cases. In two cases

the detorsion occurred at the time of the colic examination by barium enema. In the third case detorsion was effected by putting the patient in the knee chest position and placing her on right side.

The danger inherent in conservative management lies in the fact that it is difficult or at times impossible to determine when impending circulatory impairment or strangulation occurs in a strangulating volvulus. Strangulation may be suspected however if any of the following findings appear:

- 1 If the volvulus has been present for more than six hours without clinical and radiologic improvement.

- 2 If the colon proximal to the volvulus or terminal bowel is markedly distended and shows no radiologic improvement during the period of observation.

- 3 If there is a leukocytosis or elevation of temperature associated with abdominal tenderness.

Once a diagnosis of strangulation is made even contemplated conservative management should be given up and the patient promptly operated upon. In an occasional case strangulation occurs so insidiously that none of the commonly accepted criteria appear and yet necrotic bowel appears at operation. This danger must be constantly borne in mind by any surgeon electing the conservative management of this disorder. In general the safe plan of action whenever a diagnosis of volvulus of the right colon has been made is prompt surgical intervention. The only possible exception to this would be those cases of recurrent volvulus in which the obstruction is never complete and the patient passes gas per rectum.

Partial cecal volvulus may produce symptoms like those of acute appendicitis. For this reason in any case in which a diagnosis of appendicitis is made in a nonfebrile patient, volvulus of the cecum should be considered in the differential diagnosis and a survey film of the abdomen ordered.

An extremely rare cause of volvulus of the cecum was reported by Glass and Abramson. In this report volvulus of the cecum was caused by adhesions to a lithopedion. Anderson, Coulter and Woolner reviewed the literature in 1931 and found 252 cases of lithopedion reported in the

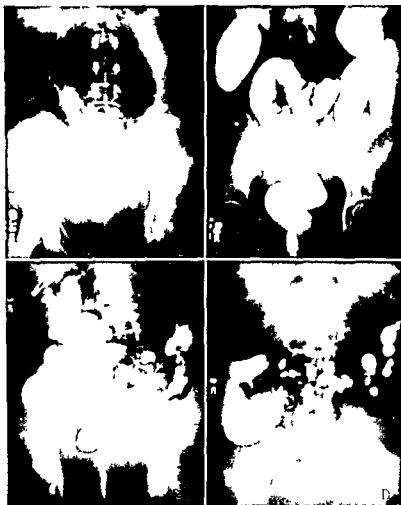


FIG. 175 A volvulus of the cecum in a postpartum patient reduced by positioning. Notice the stages in the reduction of this obstruction.

world's literature. Since the incidence of cecal volvulus is very small, the association of volvulus with lithopedion must be extremely rare.

Wilson, Desforges, Dunphy, and Campbell noted that partial obstruction of the left colon was associated with four cases of cecal volvulus in a group of eight cases of cecal volvulus studied. Three of the partial obstructions of the left colon were due to carcinoma and one was caused by a benign stricture. The mechanism involved in the production of cecal volvulus in these cases is the production of cecal distention due to the partial left colonic obstruction. This is then followed by hyperactivity. Irgiel and Ligel reported one case of cecal volvulus which occurred while the patient was under hospital observation for partial ob-

struction of the left colon just distal to the splenic flexure. The volvulus had to be surgically treated before the primary neoplastic obstructive colonic lesion could be taken care of. From these experiences it might be advisable to study any patient diagnosed as having cecal volvulus to see if a left colon obstruction is the precipitating factor. A partially obstructing carcinoma might easily be overlooked if attention is focused exclusively upon the cecal volvulus.

The prognosis in volvulus of the cecum has shown considerable improvement in the past 20 years. A mortality rate of 50 per cent was reported in 1942. Young and his associates reported seven cases with one death in 1947. Dixon and Meyer reported 13 cases with no deaths in 1948.



FIG 176 Volvulus of the cecum due to a lithopedion. An upright film of the abdomen shows fluid level in the small and large intestine. The lithopedion is visible in the right lower quadrant. This is an extremely rare type of obstruction.

Vaudenberg and Cantor have noted three cases with no deaths in 1955. This remarkable improvement in mortality rate of cecal volvulus is due to a more widespread recognition of its possible presence. The resulting early diagnosis permits adequate surgical intervention.

The transverse colon is normally not likely to undergo volvulus because it is fixed at the hepatic and splenic flexures. However in a review of 334 cases of volvulus of the colon reported by Gerwig 14 cases of volvulus of the transverse colon were reported. This 4 per cent incidence is sufficiently large to merit separate consideration. Adhesions of a long omentum to a site of previous pelvic or abdominal surgery are commonly noted as a cause for volvulus of the transverse colon. A redundant

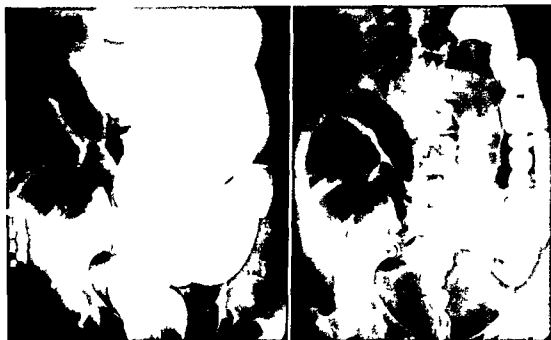
transverse colon may result in a twisting of this loop particularly if the omentum is adherent. Among the various etiologic factors reported are the following:

- 1 Elongation of the mesocolon with a mobile colon
- 2 Absence of the mesentery with a freely mobile bowel
- 3 Closely approximated points of fixation at the hepatic and splenic flexures
- 4 Congenital bands or adhesions of omentum

Volvulus of the transverse colon usually begins suddenly with the onset of cramping abdominal pain such as is found in all mechanical obstructions. Distention minimal at first later involves all loops of bowel proximal to the point of obstruction. The volvulus in this location like volvulus elsewhere is associated with the strangulating type of obstruction which is apt to occur relatively early. As a result the signs of strangulation may make an early appearance even before the onset of marked distention. The usual signs are rapid pulse, abdominal soreness, leukocytosis and shock. These depend upon the degree of circulatory impairment and are therefore variable in degree from the mildest to the most extreme.

Early in the course of this process radiologic study may show clear cut evidence of a localized distention of the affected loop of colon. There is at this time little or no distention of the small bowel. As the obstruction progresses however there may be marked distention of the small bowel and the loop of bowel involved may become fluid filled. This latter finding makes it difficult to see the transverse colon on the survey film. A barium enema may be necessary in order to confirm the presence of volvulus of the transverse colon. The contrast medium readily indicates the exact point of obstruction as well as its degree. During the barium enema volvulus of the transverse colon is occasionally reduced.

The treatment recommended for volvulus of the transverse colon is immediate surgical intervention. Simple detorsion of the obstructed loop is suggested for those cases uncomplicated by vascular impairment. Adhesions and adhesive bands binding the omentum to the pelvic structures or anterior abdominal wall should be severed or the



FIGS. 177-178. Volvulus of the transverse colon. Note the round termination of the barium at the point of obstruction and the central dentate margin. Note also in the postevacuation study the crossing and spiraling of the mucosal fold at the point of torsion in twisting volvulus.

involved omentum resected. Although the incidence of recurrence is not available for these cases of volvulus in which simple detorsion is carried out, it may be similar to that reported for sigmoid volvulus in which simple detorsion has been the operative procedure. Munro reports a recurrence rate of 20 per cent for this. These patients should be made aware of the possibility of recurrence so that when necessary medical attention will be sought promptly. For further discussion of surgical management of these lesions the reader is referred to Chapter 21.

Volvulus of the transverse colon may be chronic. Patients of this type undergo repeated attacks of partial obstruction (due to volvulus) which finally becomes complete. However, these cases are rare. At operation the involved segment of colon is found to be dilated and markedly hypertrophied. Anatomic variations of hyperfixation and points of hypofixation provide the mechanism for such volvulus. The distention in such cases may be enormous so that respiratory distress may be one of the prominent features of the process. Pain radiating to the right flank as a result of the pull of the peritoneum at the hepatic flexure some

time occurs. As a result of diaphragmatic irritation the pain may radiate to the right arm and shoulder. In a case of this type reported by River and Gubler the volvulus released itself following spinal anesthesia with the result that 5000 cc of liquid stool were passed per rectum with large amounts of gas. Nine months later this patient was operated upon for recurrent volvulus.

The third most common cause of obstruction of the left colon is volvulus. Sigmoid volvulus occurs almost three times as often as cecal volvulus and four times as frequently as small bowel volvulus. Sigmoid volvulus may occur at any age but is generally found in the older age group and is rather uncommon under 30 years of age. It is believed to occur more often in women than in men because the female pelvis is wider and the abdominal wall after pregnancy more relaxed. Because of these factors there is more room within the peritoneal cavity for the loops of bowel to rotate. Treves is of the opinion that obstinate constipation in the elderly patient is an important etiologic factor.

Volvulus is more common in the eastern European, Asiatic and African peoples because of the



FIG 179 Volvulus of the sigmoid colon. Notice the tremendously distended colonic loop. A long tube should never be used in this type of case. Immediate surgery is indicated.



FIG 180 Double closed loop obstructions. The transverse colon above and the sigmoid colon below have undergone volvulus.

course types of food eaten. In addition among the African natives and Asiatic peoples the custom of overeating after a period of fasting is believed to predispose to sigmoid volvulus. Berger and Lundberg reported four cases where sigmoid volvulus was due to lead poisoning. These authors believe that these cases involving lead plant workers with anatomically long sigmoid colons were the result of hyperactivity of the gastrointestinal tract caused by the lead poisoning. Graffenhagen suggested the possibility of a hereditary tendency toward volvulus of the sigmoid colon after having attended a father and son with the same disorder. Perlmann reported from his clinic in Russia that half of all intestinal obstruction there was caused by volvulus.

There are many conditions predisposing to the development of sigmoid volvulus. All of these act either to increase the length or diameter of the colon or tend to reduce the size of the mesenteric sigmoid attachment. It is this combination of an increased length of bowel and shortening of the mesenteric attachment that is the necessary antecedent for the development of volvulus. The

occurrence of volvulus in a megacolic segment of bowel is possibly more frequent than is realized. Weel's reviewed 63 cases of this type. He noted that half of these patients experienced previous attacks of abdominal pain, obstipation and distention which would suggest that volvulus had occurred and spontaneously reduced itself. The patients with megacolon may be classified into three large groups.

- 1 In the first group are all patients with marked dilatation of the entire colon in addition to a dilated rectum.
- 2 In this group are all those patients with severe dilatation of the colon down to the rectum which is normal.
- 3 This group accounts for those patients with enlargement of the descending and sigmoid colon with or without involvement of the rectum and proximal colon.

Congenital bands crossing the pelvic colon from right to left may occasionally produce tremendous hypertrophy and dilatation of the sigmoid colon which cause twisting. This sets the stage for volvulus. Inflammatory lesions may narrow the base of the mesosigmoid.

Sigmoid volvulus constitutes a closed loop obstruction. The pathologic changes are caused by distention of the bowel by gas and fluid. In addi-



FIG 181 Megacolon

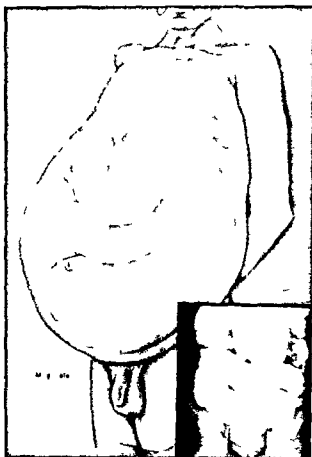


FIG 183 Appearance of a patient with a tremendous megacolon



FIG 187 Megacolon noted on a survey film

tion all cases of volvulus are potentially strangulating because of compression of the mesenteric vessels to the twisted loop of colon. The volvulus may be clockwise or counter-clockwise although the clockwise type of rotation is more common. Groth demonstrated that every volvulus of the sigmoid colon in its mesenteric axis is also associated with an axial rotation or axial torsion of the bowel itself. He postulated that the axial torsion is twice as great as the torsion of the mesentery. For this reason a mesenteric torsion of 180 degrees which is usually asymptomatic is apt to produce an axial torsion on the long axis of the colon of 360 degrees which is sufficient to produce symptoms. The extent to which the axial torsion causes stenosis depends upon whether it is evenly distributed throughout the entire sigmoid or is limited to one or more short lengths of the loop. Many of these patients have repeated attacks of

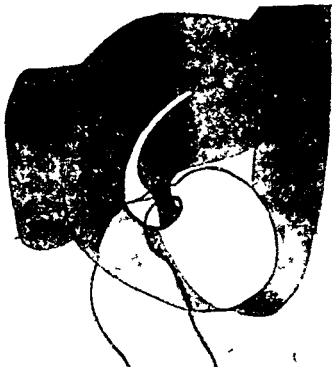
volvulus with spontaneous reduction. Each attack tends to produce further thickening of the mesentery with subsequent shortening. As a result the colon becomes increasingly elongated and the mesenteric attachment shorter and narrower so that the attacks become more frequent. With the repetition of attacks the chance of reducibility decreases and the possibility of strangulation increases. During an acute attack of volvulus of the sigmoid colon the hyperperistalsis produced by the obstruction forces air and intestinal content into the twisted sigmoid loop because the site of the torsion acts like a valve which permits air to enter but not to escape. The loop of sigmoid thus becomes markedly dilated. If there is no circulatory impairment the bowel wall remains well nourished for the first few days although it becomes progressively more distended. Because of the thickness of its wall and the anatomic distribution of intramural vessels the sigmoid can tolerate the highest pressures in the gastro intestinal tract before circulation in the wall ceases. If the circulation becomes compromised due to a twist in the mesosigmoid strangulation sets in. The condition of the patient is then much more serious. With increasing strangulation thrombosis of the mesenteric veins begins and spreads in a retrograde fashion. The strangulated loop may then become gangrenous. Perforations in the colonic wall may occur and peritonitis result. In addition the site of torsion of the colon may become damaged to such an extent that after detorsion of the sigmoid colon a perforation at the site of the torsion may occur with fatal consequences.

The symptomatology associated with acute sigmoid volvulus is variable depending upon the degree of twisting in the loop. Most of the patients appear less ill than patients with other intestinal obstructions. However Bruusgaard reported that 21 patients in his series were admitted in poor condition. Patients with strangulation of the colon may present a picture of deep shock during the first day of the disease and die on the day of admission. The most common complaint is a change in normal bowel habits or prolonged constipation. Attacks of severe cramping pain followed by diarrhea are not uncommonly reported. After many such attacks of this type the

patient may suddenly develop an acute attack which requires hospitalization. Many patients are admitted complaining of increasing abdominal distention whereas others report a progressive increase in the severity of cramping abdominal pain. In some instances a sudden sharp pain in the abdomen may be accompanied by a stoppage of the bowel movements. The pain associated with unreduced volvulus tends to be constant as a result of a pull on the mesentery but it is also accompanied by intermittent colicky pain due to hyperperistalsis. The pain of volvulus tends to localize to the region of the navel. On examination marked abdominal enlargement may be noted. Occasionally in thin walled abdomens the outline of a large intestinal loop may be apparent. Although the abdominal distention is an important feature of this disease an occasional case will appear with only a minor degree of distention. Generally the hiccorygnus associated with the bouts of colicky pain characteristic of mechanical obstruction is easily elicited. Abdominal tenderness does not usually occur unless peritoneal irritation due to strangulation is present. Vomiting on a reflex basis may appear early in the course of the disease but after this the vomiting does not generally appear until the patient is dying. The site of the volvulus is usually too high to be palpable per rectum but rectal examination reveals a large empty ampulla. The site of the volvulus is usually obvious at sigmoidoscopic examination. In many instances the spiral loop can be made out.

In the majority of cases a diagnosis of sigmoid volvulus can be established by radiologic examination. The classic case of sigmoid volvulus presents certain characteristic features. The most commonly noted radiologic features are

- 1 A markedly distended sigmoid is present
- 2 Fluid levels in the sigmoid loop show little difference from the levels in the erect position
- 3 Moderate gaseous distention of the rest of the colon is present
- 4 A balloon like distention of the cecum is seldom seen
- 5 Small bowel distention is noted only in cases associated with peritonitis or late cases of volvulus
- 6 Spiral patterns are visible on the mucous membranes at the site of the torsion



FIGS 184-185. Volvulus of the sigmoid colon. Notice the typical bird's beak shown by barium enema. Above right: Mechanism of bird's beak deformity.

7. The domes of the diaphragm are high and show limitation of motion if strangulation of bowel is present.

8. Fluid appears in the peritoneal cavity.

A barium enema generally demonstrates the characteristic outline of the proximal portion of the rectum as a bird's beak shape with a spiral narrowing.

The conservative method of reducing sigmoid volvulus was first suggested by Laurall and was first successfully performed by Nørgaard. The experience with this method of treatment became so extensive in Scandinavian countries that by 1947 Bruusgaard was able to report on 91 patients treated in this fashion. The technique of sigmoidoscopic intubation of the sigmoid colon in acute volvulus is as follows:

The patient is placed in the head down position. The sigmoidoscope is inserted carefully under direct vision. Air should not be injected into the colon. The condition of the mucosa must be carefully noted. If there is any suggestion of vascular changes operative intervention is indicated at once. By carefully inserting the sigmoidoscope the distended loop of colon may be entered. If this occurs there will be a sudden forcible evacuation of

intestinal contents through the sigmoidoscope. After decompression has been obtained a soft rubber rectal tube 28 to 30 Fr. caliber and 15 inches long should be inserted well up into the sigmoid colon. The tube should be sutured to the anus and left in position for four to five days. This is important because recurrences have been known to occur within a few hours after instrumental reduction from below. Immediately following the sigmoid intubation the abdomen should be checked by a survey film to be certain that the volvulus has been decompressed. In any case of sigmoid volvulus in which there is a possibility of vascular changes in the wall of the intestine or in which intubation was unsuccessful surgery with detorsion of the volvulus is suggested by Bruusgaard.

In a group of 91 patients treated in this fashion 168 times by Bruusgaard there was only one fatality which he could attribute to the sigmoidoscopic method of decompression. This fatality occurred when the sigmoidoscope perforated the colon. There were nine additional failures with this method; these were operated upon. Although Bruusgaard reports good results from this conservative method of treatment for acute volvulus he points out that it has no effect in the prevention of recurrences. These must be taken care of surgically.

American surgeons treat sigmoid volvulus by surgical intervention at the earliest possible moment after correction of electrolyte and water losses. At operation a simple detorsion of the twisted loop is the procedure of choice. If the viability of the bowel is questionable or if frank strangulation has occurred a resection of the colon is performed. For further discussion of surgical management the reader is referred to Chapter 21.

Recurrent volvulus has been noted by many authors. It is characterized by recurrent attacks of abdominal pain and obstipation which are relieved by diarrhea and the passage of gas after spontaneous reduction. These patients may be diagnosed as psychoneurotics because of the negative physical and radiologic findings following reduction of the volvulus. Recurrent volvulus may be grouped into three main groups:

1 In this group severe and continuous constipation with discomfort and aching in the back are the important features of the disease.

2 In this group the volvulus is characterized by the presence of constipation during the attacks of partial volvulus with freedom from constipation between attacks.

3 In this group during the attack of volvulus the pain may be very severe and the distention well marked but the torsion is not sufficiently great to completely obstruct the bowel. For this reason the bowels may move normally. Bloody diarrhea is not uncommon in this group because of vascular interference. It has been suggested that patients presenting Von Wahl's syndrome characterized by pain and diarrhea without distention may really belong to this group of volvulus.

One of the longest loops of sigmoid volvulus recorded is the case reported by Lippincott in which the sigmoid mesentery was found to be 25 cm long and the length of the distended loop of colon was 116 cm with its greatest circumference 47 cm.

In addition to volvulus upon a mesenteric axis, volvulus may result from the intertwining of two loops of bowel. This is quite rare. The sigmoid colon and terminal ileum are most commonly involved in such entanglements. Strangulation of the sigmoid colon has been reported as a result of a loop of ileum encircling the base of the sigmoid. As a result compression of the mesenteric vessels

occurred with consequent strangulation of the colon.

Intussusception

When any portion of the gastro intestinal tract invaginates into an adjacent portion the process is known as intussusception. Sixteenth century anatomists first described this obstruction process. Although the cause in many cases cannot be determined particularly in infants in adults the inciting cause is commonly found to be polyps, tumors, appendicitis, Meckel's diverticulum, foreign bodies in the bowel, trauma or acute inflammatory processes in the bowel wall. Moroni first described intussusception associated with a polyp and Meckel's diverticulum in 1896. The ileocolic type of intussusception has been reported to be responsible for the great majority of all such cases and the colocolic type of intussusception occurs next in frequency, being responsible for one eighth of the cases. The invagination usually runs in the same direction as peristalsis and the invagination is usually single. In occasional instances double or even triple intussusceptions have been reported. Retrograde intussusception has also been reported as an uncommon type. F. F. Lewis reported a case of retrograde intussusception of the pelvic colon and descending colon into the transverse colon with the apex of the intussusceptum at the cecum. Ilemming successfully operated upon an intussusception of the descending colon into the transverse colon the etiologic factor being a benign polyp. In a review of 1000 cases of intussusception of all kinds Fitzwilliams reported finding six cases of retrograde intussusception. Balfour reported actually observing the mechanism of retrograde intussusception during surgery. He believed that in the human there may be strong antiperistaltic contractions in the colon which may cause intussusception. As a result the same mechanism that is operative in the production of the usual variety of intussusception may produce retrograde intussusception in such cases. Block, Kieder and Luckstein observed such antiperistalsis radiologically.

Not all intussusceptions in the region of the right colon present the acute manifestations commonly associated with this type of obstruction. Cases of chronic intussusception of the ileocecal

OBSTRUCTION OF THE COLON

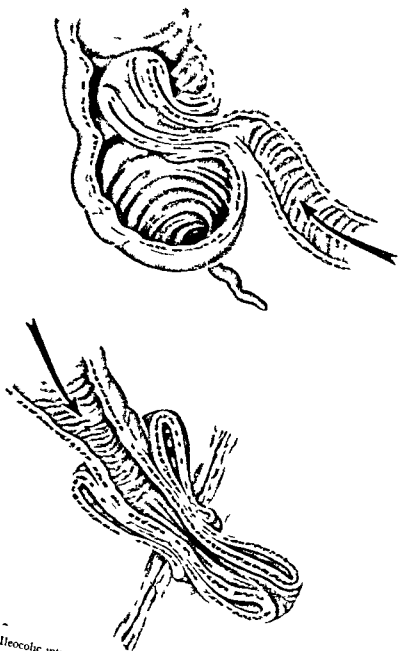


FIG 186 1861e Ileocolic intussusception Below Intussusception through a colostomy

or ileocolic variety of several years duration have been reported Goodall reviewed 21 cases of this type which lasted more than four years without complete obstruction so that normal passage of intestinal contents was possible Most of these cases were eventually operated upon On rare occasions however the spontaneous separation of

the intussusceptum resulting in a cure has been reported

The symptoms associated with intussusception depend upon the acuity of the process the completeness with which the intussusceptum fills the intussusciens and the presence of impairment of blood supply to the intussusceptum As the infold

ing process continues the mesentery of the intussusceptum becomes pulled in. The venous blood supply becomes easily obstructed or compressed. This results in an increased venous back pressure with hemorrhage, edema and finally strangulation of the intussusceptum. The first symptom usually associated with intussusception is the sudden onset of severe cramping pain in the abdomen. The pain is intermittent and is associated with hyperperistalsis such as is found in any mechanical obstruction of the ileum. The colicky pain is that of mechanical obstruction. If strangulation of the intussusceptum occurs the signs of a strangulating obstruction may be superimposed upon those of nonstrangulating obstruction. Although the bowels may move normally early in the course of the intussusception complete obstipation sets in when the obstruction becomes complete and the colon distal to the point of obstruction has been emptied. Bloody mucus is not uncommonly passed per rectum especially in those instances in which vascular compression has occurred. In those cases of ileocolic or ileocecal obstruction which have become completely obstructed marked intestinal distention becomes a prominent feature. Generally there is little tenderness or rigidity over the involved area. This is due to the fact that the ensheathed intussusceptum is covered by a normal peritoneum of the intussusciens. The presence of an abdominal tumor mass is an important finding. The older reports noted the presence of an abdominal mass associated with intussusception in half of the cases. In the more recent literature the incidence of this finding has shown an appreciable increase. The tumor mass is quite variable in shape and size. It may be sausage shaped or round. It also varies in consistency but is generally found to be soft. Ileocecal intussusceptions are usually found in the right side of the abdomen. Perrin and Lindsay report that in some instances ileocecal intussusception may reach the anus and be palpable per rectum. Laboratory studies are not helpful in arriving at a diagnosis although once a diagnosis has been made the presence of leukocytosis suggests strangulation of the intussusceptum. In the absence of strangulation the temperature and pulse are normal.

The radiologic features commonly associated with ileocecal or ileocolic intussusception are:

- 1 Presence of small bowel distention indicating mechanical obstruction when the obstruction is complete. This can be noted on a survey film.

- 2 Loss of normal gas shadow usually found in the right lower quadrant although much gas may be found on the left.

- 3 A smooth cup shaped defect produced in the colon due to the protrusion of the intussusceptum.

- 4 If the obstruction is incomplete some barium may pass between the intussusceptum and the ensheathing intussusciens.

- 5 Radiologic study may reveal a distal progression of the obstructing process.

- 6 The intussusceptum may be pushed back or entirely reduced during the fluoroscopic manipulation.

- 7 The tumor inciting the adult type of intussusception may be radiographically demonstrated.

The transverse colon not infrequently is obstructed by ileocecal or ileocolic intussusception by virtue of the intussusceptum passing distally to this point. Colocolic intussusceptions have been reported at the anus with the neck of the process at the transverse colon.

Acute sigmoidorectal intussusception is rather uncommon. It can easily be diagnosed by a sigmoidoscopic examination. This not only provides a diagnosis of the type of obstruction present but also corrects the intussusception. The obturator of the sigmoidoscope usually begins the reduction of the intussusception which is completed by the insufflation process. Among the unusual and rare varieties of intussusception is the occasional report of intussusception of the terminal ileum and cecum through the ascending colon which finally present itself at the anus. Gangrenous appendices have been reported protruding from the anus as a result of such an intussusceptive process. The rarity of these cases makes them medical curiosities. That they can occur however attests to the distance that an intussusception may pass through the gastrointestinal tract. The retrograde intussusception which may occasionally obstruct the left colon is discussed earlier in this section.

Diverticulitis

Diverticulitis of the cecum and the right colon are uncommon and are rarely diagnosed correctly prior to surgery. The incidence of cecal and right colon diverticulum has been variously reported as constituting from 15 to 6 per cent of cases. There appears to be considerable controversy over the etiology of this lesion. There have been various mechanisms proposed to explain the development of a diverticulum in the right colon. These range from traction on the cecum wall due to postoperative adhesions to the concept that such diverticula are congenital. This latter belief is the most commonly accepted. Such diverticula are classified with the true bowel diverticulum and their walls contain all layers.

Cecal diverticulitis may occur at any age. The youngest case reported was 3 years of age and the oldest 69. The disorder appears to be almost evenly divided between the sexes although colonic diverticula in general are more common in males in a ratio of two to one. The type of obstruction associated with diverticulitis of the right colon varies. The acute inflammatory process may cause paralytic ileus like any other intra abdominal inflammatory process. Perforation of the diverticulum may result in a generalized peritonitis with its ileus or more commonly the perforation may be come walled-off with the formation of an abscess. This may cause a mechanical obstruction by compression or as a result of adherence of a loop of small bowel to the abscess. This process of mechanical obstruction may occur during or after the paralytic ileus associated with the intra abdominal inflammatory process.

The signs and symptoms associated with this process are those generally found with any intra abdominal inflammation. Pain in the abdomen, nausea, vomiting, and later distention are most commonly found. It may be difficult or impossible to differentiate between cecal diverticulitis and acute appendicitis. Slowly perforating cecal or right colon diverticulitis with the formation of a chronic granuloma may be confused with carcinoma of the right colon.

Conservative therapy with intestinal intubation using the long intestinal decompression tube and

antibiotics will successfully correct most cases of acute diverticulitis of the right colon. If the abdomen is opened it may be difficult to differentiate cecal diverticulitis from carcinoma. A biopsy is helpful in such cases. A diversionary ileocolostomy is an adequate operation for the inflammatory process. A study should be carried out to determine whether resection of the right colon is indicated in multiple diverticula with extensive scarring of bowel. Such cases are uncommon.

The prognosis for cecal diverticulitis is good. An over all mortality of 58 per cent has been reported in the literature reviewed by Vaughan and Varsete.

Diverticulitis is most commonly found in the left colon. Approximately three-quarters of all such diverticula have been reported as being found in the sigmoid colon. Diverticulitis may pro-



FIG 187 Diverticulitis of the descending colon with obstruction

duce intestinal obstruction in many different ways. The following mechanisms may occur:

1. Acute diverticulitis (sigmoid) may produce an inflammatory swelling with the concomitant acute intestinal obstruction.

2. Diverticulitis may lead to perforation forming an abscess which causes paralytic ileus. At times the abscess may reach such proportions that it mechanically obstructs the colon by pressure or edema in its contiguous wall. In such cases the paralytic ileus may gradually merge into a mechanical obstruction.

3. Upon the resolution of the inflammatory process, the resultant fibrotic changes in the colon may cause chronic intestinal obstruction.

4. Acute diverticulitis, with or without perforation and abscess, may be present with concomitant

carcinoma of the colon. This possibility must be considered. The importance of this lies in the marked difference in treatment for these two obstructive processes.

Acute diverticulitis requires conservative management. The presence of a superimposed acute colonic obstruction, with or without perforation and abscess formation, necessitates a complete de-functionizing colostomy. The surgical management is discussed in detail in Chapter 21.

Occlusion of Blood Vessels

Strangulation of the right colon as a result of occlusion of the blood supply may be classified as primary or secondary. The primary type occurs when arterial or venous obstruction to the mesenteric artery is sufficiently high to involve the right colic, middle colic or ileocolic vessels. Thrombosis or occlusion of the superior mesenteric artery at its point of origin results in strangulation of the



FIG. 188 Diverticulitis of the descending colon with obstruction (from the patient in Figure 187)



FIG. 189 Obstruction of the left colon. Small bowel distention has not occurred as yet. Note that the right colon and transverse colon are filled with feces and as a result the contrast of gaseous distention is not noticeable.

entire right colon in addition to the strangulation of the small bowel. The same etiologic factors causing occlusive mesenteric vascular disease of the small bowel are also operative in the right colon. The secondary type of occlusive vascular disease of the right colon is that found secondary to cecal volvulus or intussusception in which the primary lesion is mechanical obstruction and the mesocolonic vessels become secondarily obstructed.

The signs and symptoms of mesocolonic vascular occlusive disease cannot be distinguished from those found with the vascular occlusive disease of the small bowel. In the primary occlusive group a diagnosis of colonic involvement is seldom

made prior to surgery. In the secondary occlusive vascular group recognition of the type of obstruction present when coupled with signs of strangulation facilitates the diagnosis.

The mortality rate for the group of primary occlusive diseases of the right colon is high because the superior mesenteric artery is obstructed so high that the entire small bowel and colon are apt to become gangrenous. In an occasional case the right colic or middle colic branches may be embolically obstructed so that primary resection and anastomosis may be successfully carried out. In general the outlook for this primary group is dismal. Early surgical intervention offers the only hope for a successful outcome yet early diagnosis is difficult.

Occlusive vascular disease involving the inferior mesenteric artery is rare. As a result the left colon rarely becomes involved in strangulating obstructions of this type. For an amplified discus-

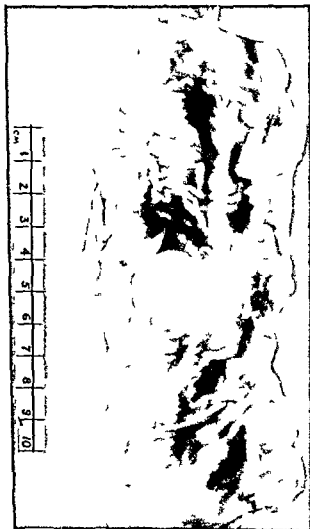


FIG 190 Endometriosis of the descending colon as a result of extension from the left ovary with obstruction



FIG 191 Idiopathic megacolon

sion the reader is referred to the section on occlusive vascular disease in Chapter 8

Internal Hernia

This is a rare cause of colonic obstruction. Derangements in intestinal rotation may create conditions so that the right colon is found obstructed in right paraduodenal fossa hernia. Cases of this type as well as the retroposition of the transverse colon are almost medical curiosities. Rose in reporting a case of his own reviewed the literature from 1883 through 1940 and found only 12 similar cases reported. The seriousness of this condition is indicated by the fact that 7 of the 12 patients died. If the condition is suspected and carefully looked for a correct radiologic diagnosis can be made. Prompt surgical intervention offers the only hope.

Hamaker reported a rare type of obstruction of the transverse colon. In this case the colon and omentum had prolapsed through a hole in the upper portion of the small bowel mesentery.

The splenic flexure is not infrequently involved

in a diaphragmatic hernia. It may become obstructed in such hernial processes. This is most likely to occur in a traumatic diaphragmatic hernia. The management of this type of case is surgical. Incarceration of the splenic flexure in a diaphragmatic hernia may be erroneously diagnosed as obstructing carcinoma of the proximal left colon. A case of this type follows.

O. C. white male age 39 was admitted to Grace Hospital with a diagnosis of bowel obstruction. An X-ray on admission disclosed a stenosing lesion in the proximal descending colon near its junction with the splenic flexure. The lesion measured 1 inch in length. A chest X-ray taken at the same time disclosed an elevation of the right leaf of the diaphragm and a recent minimal pleural effusion in the left lower chest suggesting the possibility of metastasis. At operation a hernia of the left leaf of the diaphragm was found containing 14 inches of colon.

Turel reported a case in which the sigmoid colon herniated through a hole in the mesosigmoid and became strangulated. The hole in the mesosigmoid was between 3 and 4 inches long and ex-



FIG. 192. Megacolon in a horse.



FIG. 193. This is a good example of high grade partial obstruction of the colon and transverse colon due to fecal impaction.

tended from the attachment of the mesosigmoid to the mesenteric border of the bowel. Its edges were smooth rounded and thickened. Turell believed that this was of long standing and probably congenital in origin. Turell reduced the herniated sigmoid which then regained its normal luster and peristaltic activity. The rent in the mesocolon was closed by interrupted sutures.

Endometriosis

Intestinal obstruction as a result of endometriosis has been frequently observed. Although chronic obstruction of the colon from endometriosis is relatively common, acute intestinal obstruction from this source is not. Because of their pelvic position the sigmoid, rectosigmoid and rectum are the most common sites of endometrial bowel implantation. The sigmoidal lesions of endometriosis producing obstruction present three varied mechanisms:

1. The lesion may cause a concentric scarring of the bowel wall due to the infiltrating endometriosis.

2. There may be a polypoid submucosal infiltration obstructing the lumen by its mass.

3. The excentric scarring, caused by the endometriosis may cause acute angulation of the bowel obstructing it.

The endometrial implants on the colon like those elsewhere depend upon the hormonal ovarian influence. The endometrial implants undergo retrogression in the absence of ovarian hormonal stimulation. However, once fibrosis and scarring have occurred this remains as a permanent obstruction. Usually endometriosis is found in women during the childbearing age. Most of these are either sterile or have become so after the birth of one child. An important clue to the clinical diagnosis is the fact that the recurrence of symptoms usually varies with the menstrual cycle. However, unlike endometriosis of the ileum which occurs in young women, endometriosis of the colon occurs in older women and is generally a condition of many years duration.

The treatment for this type of obstruction depends upon the extent of pelvic involvement, the age of the patient, her desire to bear children or her ability to do so determined by examination of

her pelvic organs at the time of surgery and the degree of scarring present. The decision as to whether or not to do a bilateral oophorectomy must be based upon these factors. Generally in young women with little pelvic involvement resection of the obstructed bowel might be the best procedure since this would permit childbearing. In older women beyond the childbearing age or in younger women with extensive involvement of the pelvic viscera a bilateral oophorectomy and hysterectomy may be required. In these cases the bowel should be treated conservatively and extirpation of the hormonal influence should be the treatment of choice.

The possibility of carcinoma developing from an endometriosis is still controversial. Although some doctors report a presumable relationship there is no absolute proof that it exists.

Koentgen therapy in the treatment of endometriosis is reserved for those patients for whom



FIG 194 The same patient as in Figure 193. Fecal impaction producing high grade partial colonic obstruction. This film demonstrates a normal colon following numerous enemas and thorough gastro-intestinal cleansing.

a diagnosis has been established who are poor surgical risks or who have an intestinal recurrence of endometriosis following conservative surgical management

Ulcerative Colitis with Stenosis

The lumen of the colon may become obstructed as a result of stenosis produced by the formation of hyperplastic granulomatous tissue due to ulcerative colitis. In such cases the patient may show signs of obstruction such as intermittent intestinal distention along with such symptoms of colitis as diarrhea, tenesmus, blood and mucus in the stool. Many of these cases respond dramatically to injections of cortisone and ACTH. The granulomatous areas involute with such treatment. A typical example of this type is the following:

L.K., a white female, age 32, was admitted to the hospital with a diagnosis of high grade partial intestinal

obstruction. She proved to have had ulcerative colitis a year before. During this time she had 10 to 15 watery stools daily with considerable blood and mucus. During the month prior to admission she had noted that despite the frequency of bowel movements she was becoming distended. Associated with this distention were cramping pains in the abdomen which were intermittent. Bowel movements relieved the pain. Just prior to admission a tumor mass was noted in the left lower quadrant. This was painless and movable. The patient was moderately distended. Barium enema disclosed the typical findings of ulcerative colitis with an area of marked narrowing and partial sigmoid obstruction. ACTH was started. The tumor mass began to regress almost immediately. Within two weeks it had almost completely disappeared. With the disappearance of the mass all signs and symptoms of obstruction disappeared.

As an aftermath of such granulomatous hyperplastic areas in the left colon, scarring may occur in the wall of the bowel. This results in the production of fibrotic stenotic areas which are of a



FIG 195 This is a good example of fecal impaction producing colonic obstruction. Note the gas distended colon and the pearly appearance of the greatly distended rectum as well as the cecum filled with fecal material.

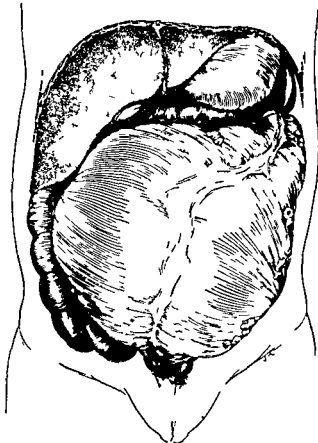


FIG 196 An enormously dilated descending colon filled with fecal material.

permanent nature. Marshall, Lester and Friedman reported a case of this type. In this case a mega-colon was found as a complication of the ulcerative colitis. The area of narrowing in the sigmoid was thought to be insufficient to cause the mega-

colon on a purely mechanical basis. They suggested that the colon (the sigmoid and lower descending colon) might have become the site of a neural histopathology which resulted in the development of a mega-colon because of functional obstruction.

Neurogenic Colonic Obstruction

Ogilvie, Dunlop and Handley have each reported cases of false colonic obstruction. The cause of the colonic obstruction in these cases was found to be an invasion of the celiac plexus by malignant cells. These authors believe that this syndrome is a definite clinical entity. In all the cases reported the clinical and physical findings were indistinguishable from mechanical obstruc-

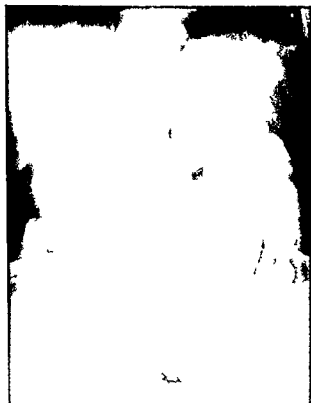


FIG 197 Patient I. S. age 56. Hysterectomy 10 years previously followed by fistula and abscess. Six surgical procedures to correct the fistula. During one of these the distal sigmoid was completely transected. In addition the patient presented an obstruction of the terminal ileum. This had been short circuited by ileocolostomy. Note the complete break in continuity of the colon at the recto-sigmoid. This was corrected by resection of the terminal ileum, cecum and part of the ascending colon followed by an end-to-end anastomosis of the ileum to the remaining portion of the ascending colon. A resection of the obstructed and dilated rectosigmoid was then performed. This was followed by abdomino transsacral anastomosis between the descending colon and the mid rectum. A defunctionizing colostomy was performed at the hepatic flexure because the rectal anastomosis was snug. The patient made an uneventful recovery and when last seen had normal bowel habits. This is a good example of what can be accomplished by careful study. It also indicates that in any patient obstructed following surgery the etiology of the obstructing mechanism may be multiple.



FIG 198 Obstruction of the lower sigmoid due to direct extension from carcinoma of the prostate producing narrowing at this point. In addition the patient presented a large polyp just below the point of obstruction. As a result although the bowels could be evacuated from above the introduction of barium from below caused the polyp to obstruct the narrowed sigmoid colon giving rise to this characteristic appearance. This might be confused with a completely obstructed primary carcinoma of the colon.

a diagnosis has been established who are poor surgical risks, or who have an intestinal recurrence of endometriosis following conservative surgical management

Ulcerative Colitis with Stenosis

The lumen of the colon may become obstructed as a result of stenosis produced by the formation of hyperplastic granulomatous tissue due to ulcerative colitis. In such cases the patient may show signs of obstruction such as intermittent intestinal distention along with such symptoms of colitis as diarrhea, tenesmus, blood and mucus in the stool. Many of these cases respond dramatically to injections of cortisone and ACTH. The granulomatous areas involute with such treatment. A typical example of this type is the following:

I K, a white female, age 32, was admitted to the hospital with a diagnosis of high grade partial intestinal

obstruction. She proved to have had ulcerative colitis a year before. During this time she had 10 to 15 watery stools daily with considerable blood and mucus. During the month prior to admission she had noted that despite the frequency of bowel movements she was becoming distended. Associated with this distention were cramping pains in the abdomen which were intermittent. Bowel movements relieved the pain. Just prior to admission a tumor mass was noted in the left lower quadrant. This was painless and movable. The patient was moderately distended. Barium enema disclosed the typical findings of ulcerative colitis with an area of marked narrowing and partial sigmoid obstruction. ACTH was started. The tumor mass began to regress almost immediately. Within two weeks it had almost completely disappeared. With the disappearance of the mass all signs and symptoms of obstruction disappeared.

As an aftermath of such granulomatous hyperplastic areas in the left colon, scarring may occur in the wall of the bowel. This results in the production of fibrotic stenotic areas which are of a



FIG. 195 This is a good example of fecal impaction producing colonic obstruction. Note the gas distended colon and the puffy appearance of the greatly distended rectum as well as the rectum filled with fecal material.

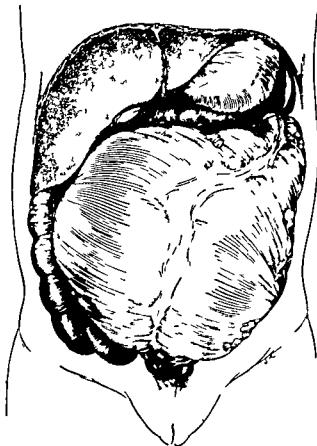


FIG. 196 An enormously dilated descending colon filled with fecal material.

permanent nature. Marink Lester and Friedman reported a case of this type. In this case a mega colon was found as a complication of the ulcerative colitis. The area of narrowing in the sigmoid was thought to be insufficient to cause the mega

colon on a purely mechanical basis. They suggested that the colon, the sigmoid and lower descending colon might have become the site of a neural histopathology which resulted in the development of a megacolon because of functional obstruction.

Neurogenic Colonic Obstruction

Ogilvie Dunlop and Handley have each reported cases of false colonic obstruction. The cause of the colonic obstruction in these cases was found to be an invasion of the celiac plexus by malignant cells. These authors believe that this syndrome is a definite clinical entity. In all the cases reported the clinical and physical findings were indistinguishable from mechanical obstruc-

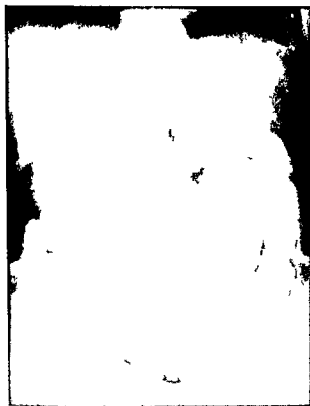


FIG 197 Patient L. S. age 56. Hysterectomy 10 years previously followed by fistula and abscess. Six surgical procedures to correct the fistula. During one of these the distal sigmoid was completely transected. In addition the patient presented an obstruction of the terminal ileum. This had been short-circuited by ileocolotomy. Note the complete break in continuity of the colon at the recto-sigmoid. This was corrected by resection of the terminal ileum, cecum and part of the ascending colon followed by an end-to-end anastomosis of the ileum to the remaining portion of the ascending colon. A resection of the obstructed and divided recto-sigmoid was then performed. This was followed by abdomino-transsacral anastomosis between the descending colon and the mid rectum. A defunctionizing colostomy was performed at the hepatic flexure because the rectal anastomosis was snug. The patient made an uneventful recovery and when last seen had normal bowel habits. This is a good example of what can be accomplished by careful study. It also indicates that in any patient obstructed following surgery the etiology of the obstructing mechanism may be multiple.



FIG 198 Obstruction of the lower sigmoid due to direct extension from carcinoma of the prostate producing narrowing at this point. In addition the patient presented a large polyp just below the point of obstruction. As a result, although the bowels could be evacuated from above the introduction of barium from below caused the polyp to obstruct the narrowed sigmoid colon giving rise to this characteristic appearance. This might be confused with a completely obstructed primary carcinoma of the colon.

GASTROINTESTINAL OBSTRUCTION

a diagnosis has been established who are poor surgical risks or who have an intestinal recurrence of endometriosis following conservative surgical management

Ulcerative Colitis with Stenosis

The lumen of the colon may become obstructed as a result of stenosis produced by the formation of hyperplastic granulomatous tissue due to ulcerative colitis. In such cases the patient may show signs of obstruction such as intermittent intestinal distention along with such symptoms of colitis as diarrhea, tenesmus, blood and mucus in the stool. Many of these cases respond dramatically to injections of cortisone and ACTH. The granulomatous areas involute with such treatment. A typical example of this type is the following:

L.K., a white female age 32 was admitted to the hospital with a diagnosis of high grade partial intestinal

obstruction. She proved to have had ulcerative colitis a year before. During this time she had 10 to 15 watery stools daily with considerable blood and mucus. During the month prior to admission she had noted that despite the frequency of bowel movements she was becoming distended. Associated with this distention were cramping pains in the abdomen which were intermittent. Bowel movements relieved the pain. Just prior to admission a tumor mass was noted in the left lower quadrant. This was painless and movable. The patient was moderately distended. Barium enema disclosed the typical findings of ulcerative colitis with an area of marked narrowing and partial sigmoid obstruction. ACTH was started. The tumor mass began to regress almost immediately. Within two weeks it had almost completely disappeared. With the disappearance of the mass all signs and symptoms of obstruction disappeared.

As an aftermath of such granulomatous hyperplastic areas in the left colon, scarring may occur in the wall of the bowel. This results in the production of fibrotic stenotic areas which are of a



FIG 195 This is a good example of fecal impaction producing chronic obstruction. Note the greatly distended colon and the pebbly appearance of the greatly distended rectum as well as the cecum filled with fecal material.

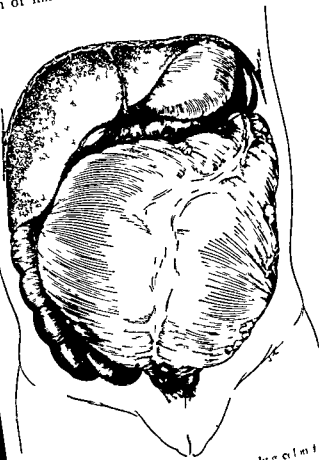


FIG 196 An enormously distended cecum filled with fecal material.

permanent nature. Marink Lester and Friedman reported a case of this type. In this case a mega colon was found as a complication of the ulcerative colitis. The area of narrowing in the sigmoid was thought to be insufficient to cause the mega

colon on a purely mechanical basis. They suggested that the colon, the sigmoid and lower descending colon might have become the site of a neural histopathology which resulted in the development of a megacolon because of functional obstruction.

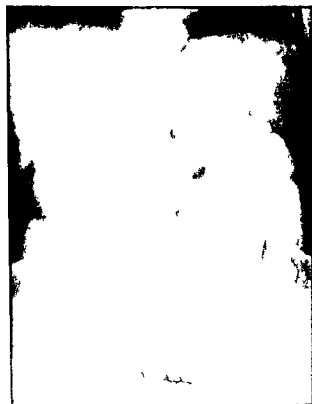


FIG 197 Patient L. S. age 56. Hysterectomy 10 years previously followed by fistula and abscess. Six surgical procedures to correct the fistula. During one of these the distal sigmoid was completely transected. In addition the patient presented an obstruction of the terminal ileum. This had been short-circuited by ileocolostomy. Note the complete break in continuity of the colon at the recto-sigmoid. This was corrected by resection of the terminal ileum, cecum and part of the ascending colon followed by an end-to-end anastomosis of the ileum to the remaining portion of the ascending colon. A resection of the obstructed and divided rectosigmoid was then performed. This was followed by abdomino-tran sacral anastomosis between the descending colon and the mid rectum. A defunctionizing colostomy was performed at the hepatic flexure because the rectal anastomosis was snug. The patient made an uneventful recovery and when last seen had normal bowel habits. This is a good example of what can be accomplished by careful study. It also indicates that in any patient obstructed following surgery the etiology of the obstructing mechanism may be multiple.

Neurogenic Colonic Obstruction

Ogilvie, Dunlop and Handley have each reported cases of false colonic obstruction. The cause of the colonic obstruction in these cases was found to be an invasion of the celiac plexus by malignant cells. These authors believe that this syndrome is a definite clinical entity. In all the cases reported the clinical and physical findings were indistinguishable from mechanical obstruction.



FIG 198 Obstruction of the lower sigmoid due to direct extension from carcinoma of the prostate producing narrowing at this point. In addition the patient presented a large polyp just below the point of obstruction. As a result although the bowels could be evacuated from above the introduction of barium from below caused the polyp to obstruct the narrowed sigmoid colon giving rise to the characteristic appearance. This might be confused with a completely obstructed primary carcinoma of the colon.

GASTROINTESTINAL OBSTRUCTION

tion of the colon. In all cases no abnormality in the colon aside from dilated bowel could be found at operation. The basic factor responsible for the syndrome was considered to be a spastic persistent ileus.

Zimmerman reported a persistent spastic ileus which resulted in colonic obstruction. The literature supports the view that when spastic ileus persists long enough it can produce a complete picture of mechanical obstruction including the distention. Many causes have been proposed. Among these are (1) stimuli acting upon the bowel wall directly (2) stimuli reaching the bowel from a distance (3) stimuli reaching the bowel from the central nervous system (4) unknown causes possibly psychosomatic.

The ganglionic segments of colon characteristic of Hirschsprung's disease are well known neurogenic causes of colonic obstruction. Lacking peristalsis this segment of bowel obstructs the normal evacuation of feces and gas. This entity is presented in detail in the chapter dealing with obstruction in infancy and childhood (Chapter 13).

Fecal Impaction

Tremendous colonic distention reaching huge proportions may occur as a result of fecal impaction. However cases of this type are uncommon.

Injuries to Colon

Injuries to the rectosigmoid at the time of surgery may result in complete obstruction at this point. This is due to the fibrosis which results from the healing of the injured bowel. The resolution of the pericolic abscess, which so frequently follows injury to the rectosigmoid is a source of considerable fibroblastic proliferation. This may eventually seal the rectosigmoid thus obstructing it.

Polyps

Pedunculated polyps of large size may produce a ball valve type of colonic obstruction. In cases of this kind normal evacuation occurs from above but enemata or barium cannot be introduced from below because by so doing the liquid stream pushes the polyp against the narrowed rectosigmoid obstructing it.

10

OBSTRUCTION OF THE RECTUM AND ANUS

With the exception of the rectosigmoid area which is actually a constricted portion of the left colon (see Chapter 9) the rectum adapted by nature for storage does not lend itself to obstruction. Below the rectosigmoid area the rectum is so ample in its luminal diameter that obstructions are rather uncommon except for those due to congenital anomalies. On occasion however the rectum does become the seat of an obstructive process. Although this is usually not complete it may sometimes become so.

INFECTIONS

Inflammatory lesions involving the rectal mucosa may cause obstruction. At rare intervals extrinsic inflammatory lesions may produce stenosis of the rectum without mucosal involvement. Gerwig reported a case of extrinsic annular fibrosing stenosis of the rectum causing obstruction which was the result of a pelvic abscess following an appendectomy for a chronic appendix. The abscess was drained for one year. Thirty two years later the patient returned with signs of intestinal obstruction. At operation the obstruction was found to be due to a marked fibrotic constriction of the rectum 8 cm. in length with encircling rings of scar tissue 0.5 cm. in thickness. It was believed by Gerwig that this area of fibrosis was the result of a long standing pelvic infection which had followed the appendectomy. Microscopic sections of this fibrotic ring of tissue showed chronic inflammatory tissue without specific infection.

A tumor mass in the rectum should not immediately be diagnosed as a malignant lesion. In an occasional case infection with *Entamoeba histolytica* may produce a hyperplastic mass with or

without abscess formation which may result in a high grade partial obstruction. Biopsy of such areas will facilitate a correct diagnosis. In general these inflammatory lesions respond to conservative management.

Among the more uncommon infections involving the rectum and causing obstruction is granuloma inguinale. This may be long standing ultimately producing a stenotic process in the rectum.



FIG 199 Granuloma of the rectum due to granuloma inguinale. Treatment was abdomino-transsacral resection and end to end anastomosis.

GASTRO INTESTINAL OBSTRUCTION

just above the anus. In cases of this type the obstruction is chronic in nature and becomes progressively worse. An acute obstruction may develop at this stenotic area as a result of fecal impaction or ingested foreign bodies which can cause such high grade chronic obstructions to become acute. Occasionally enteroliths or gallstones may become impacted at this site of narrowing and completely obstruct the rectum.

Ulcerative colitis may at times be associated with granuloma formation of such size and with such a narrowing effect upon the rectal lumen that stenosis and intestinal obstruction occur. In such cases there is a long history of ulcerative colitis with progressively increasing fibrosis which finally leads to acute obstruction.

These granulomatous obstructions can usually be treated by conservative measures. Defunctionizing transverse colostomy gives the involved area an adequate rest permitting healing to occur. Such granulomatous masses also respond well to the use of ACTH and cortisone. When the lesion has become obstructive because of a fibrosing creatrizing process a combined abdomino transsacral type of operation with resection of the involved area of rectum may be used. In such cases an end to end anastomosis is performed at a very

low level thus preserving sphincteric function. In the type of obstruction reported by Gerwig the treatment successfully used was planned after the Fredet Ramstedt operation. A sigmoidoscope was passed from below. Then a rectal tube was passed up into the rectum through the stenosed portion until it had passed through the stenosed portion of the rectum. The rectum was then mobilized and the annular constricting rings of scar tissue were cut up to the mucosa which was permitted to bulge through much as it does in the Ramstedt procedure. By extending the incision through the scar tissue along the entire length of the obstructing bands it was possible to create a bulge of the rectal mucosa throughout the entire length of the stenotic area. A follow up of this patient showed complete relief of all obstructive symptoms.

FOREIGN BODIES

Numerous foreign bodies have been removed from the rectum to relieve an obstruction. Among these are snuff boxes, drinking glasses, soft drink bottles, tin cups, preserve pots, beer glasses, mortar pestles, stones, sticks of wood, candle boxes and bullock's horn. One case was reported in which a quart of watermelon seeds was removed from the rectum where it had produced complete intestinal obstruction.

These foreign bodies were inserted for many reasons. In some instances the foreign body was inserted in the course of an act of perversion. In other instances the foreign material was introduced by the patient to correct rectal prolapse or to check diarrhea.

POSTOPERATIVE CAUSES

Rectal injury during the course of pelvic surgery is not uncommon. This is especially apt to occur in the presence of endometriosis characterized by dense adhesions or during extensive dissection in the course of operative procedures for widespread pelvic inflammatory disease. As a result of long standing infection a tenosynovitis, creatrizing obstructing process may involve the rectum. A typical case follows.

M. McC., age 37, white female was admitted to Grace Hospital December 6, 1943, with a diagnosis of intestinal obstruction. An examination disclosed a stenotic area in the upper rectum as a result of a fistula which had de-

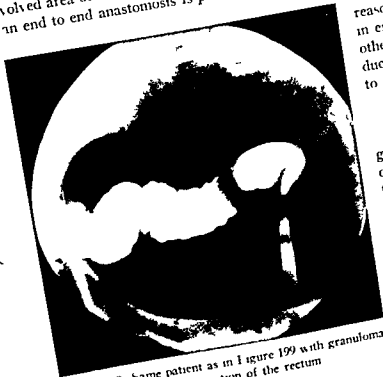


FIG. 200 Same patient as in Figure 199 with granuloma in anquale producing obstruction of the rectum

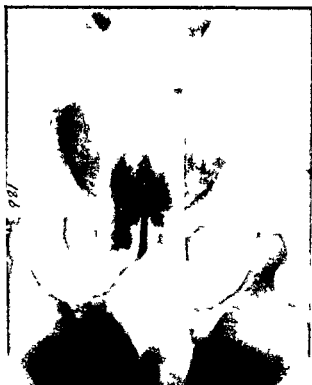


FIG 201 Foreign body in the lower rectum producing obstruction. This foreign body was removed by colotomy since it was impossible to remove it from below.



FIG 202 A pop bottle in the rectum producing obstruction. This was removed from below under anesthesia.

veloped between the rectum and the cervical stump following a supracervical hysterectomy. Microscopic examination of the rectal wall demonstrated extensive fibrosis present in the submucosal areas and throughout the entire structure of the rectal wall. The mucosa showed areas of ulceration with granulation tissue formation.

CONGENITAL ANOMALIES

Congenital rectal and anal anomalies causing obstruction are presented in detail in the chapter dealing with obstruction in infancy and childhood (Chapter 13).

PRESSURE OF PELVIC TUMORS OR MASSES

Large fibroid tumors, large ovarian tumors or cysts, and extensive endometriosis may produce obstruction of the rectum due to compression. The rectum may be simply compressed against the bony pelvis by the size of the pelvic tumor mass. The treatment which immediately relieves rectal obstruction in all such cases is simply removal of the pelvic tumor. In some cases, in order to decompress the colon it may be necessary prior to

surgery to thread a rectal tube up into the sigmoid proximal to the point of compression. This can usually be done by the use of a sigmoidoscope with the patient in the knee chest position. In this position the pelvic tumor falls forward away from the rectum, thus permitting the upward passage of the rectal tube.

NEOPLASMS

Although neoplasms seldom cause complete obstruction of the rectum below the rectosigmoid, partial obstruction is relatively common. The size of the rectum is such that the tumor would have to be very large before complete obstruction could occur. Of the neoplasms causing obstruction of the rectum, carcinoma is the most common, while sarcoma is rare. Carcinoma of the rectosigmoid, which is a particularly common cause for obstruction, is discussed with the sigmoid colon (Chapter 9). Large polypoid tumors just below the rectosigmoid may obstruct the rectum by intussusception or as a result of a fecal impaction in the rectum narrowed by the tumor.

GASTRO INTESTINAL OBSTRUCTION

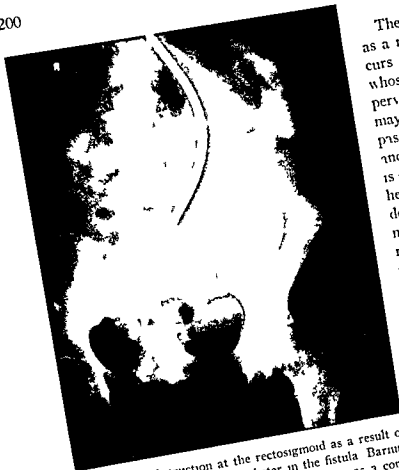


FIG. 203 Obstruction at the rectosigmoid as a result of previous surgery. Note the catheter in the fistula. Barium injection visualizes the sigmoid colon. There was a complete separation of the colon at this point. Treatment was resection of the obstructed colon and end to end anastomosis. Recovery was uneventful.

Rectal neoplasms are usually diagnosed long before acute obstruction occurs. Benign, within the finger and sigmoidoscope range of the examining physician lesions in this portion of the gastrointestinal tract lend themselves to early diagnosis and treatment. An occasional case of late neglected undiagnosed rectal obstruction due to carcinoma may require a defunctionizing colostomy as a preliminary procedure to surgical extirpation of the lesion itself. Since in most cases the obstruction is incomplete and generally below the peritoneal reflexion, abdominoperineal resection is the surgical procedure of choice. In this event sigmoid colostomy adequately decompresses the colon at the time of surgery. In any case in which an anterior resection is decided upon as the operative procedure a preliminary defunctionizing transverse colostomy is desirable.

FECAL IMPACTION

The rectum is very often completely obstructed as a result of fecal impaction. This generally occurs in aged and debilitated bedridden patients whose physiologic functions are not carefully supervised. The mass of fecal material in the rectum may become so large that virtually nothing can pass through it. These impactions are usually soft and putty like so that an effective seal to excretion is created. The chronically ill patient with cerebral hemorrhage or multiple sclerosis not uncommonly develops such fecal impactions. Nothing short of manual removal of the fecal material followed by repeated and copious enemata will suffice to remove the obstruction.

X RAY IRRADIATION

Strictures of the rectum may occur following radium and X ray treatment for carcinoma of the cervix. In such cases the stenosis may reach such a degree that only a very small lumen remains. The proctitis resulting from the X ray and radium is responsible for this type of obstructive process. Although this sort of obstruction is incomplete it is a very disabling lesion in that it often produces a bloody diarrhea. The tenesmus associated with this causes severe discomfort. Defunctionizing transverse colostomy results in immediate decompression and permits the severely inflamed rectum to quiet down. The fibrosis, however, often results in a permanent stenosed rectum so that the colostomy cannot be closed. In most cases because of the extensive scar tissue formation in the pelvis of the stenosed rectum is not possible because of the extensive scar tissue formation in the pelvis. In addition one can never be certain whether the cervical carcinoma has been cured or simply arrested. In this latter event further irradiation would be required should symptoms return. If an exenteration type of operation is decided upon the presence of a transverse defunctionizing colostomy simplifies the surgical procedure.

METASTATIC GROWTHS

Metastatic carcinoma from the cervix can produce obstruction of the rectum. Direct involvement of the rectum by infiltration of its wall is not uncommon as a result of carcinoma of the cervix.

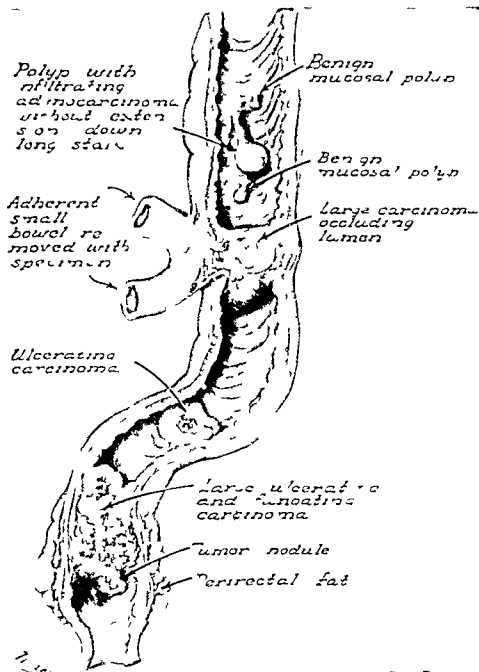


FIG. 204 Multiple primary carcinomas of the rectum causing obstruction associated with multiple benign polyps (from Peters and Eckman)



FIG. 205 Tremendous fecal impaction in the rectum. Notice the pebbly appearance.



FIG. 207 Stenosing proctitis following radium and X-ray irradiation for carcinoma of the cervix.



FIG. 208 Note the large fecaloma in the rectal ampulla. This caused intestinal obstruction.

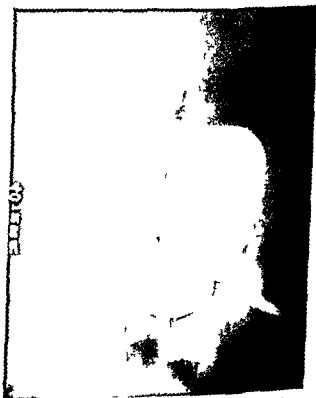


FIG. 209 Fecal obstruction caused by metastasis from carcinoma of the cervix.

A similar type of invasion may occur with carcinoma of the ovary. In such cases the rectal involvement is usually a terminal process so that although ureteral obstruction and uremia is the most common cause of death in carcinoma of the cervix the intestinal obstruction may become a terminal factor. In such case defunctionizing colostomy adequately decompresses the colon. No attempt should be made to treat the rectal obstruction itself. In any case of this type one should be certain that the small bowel is not involved with the rectum in this neoplastic invasion. When this occurs defunctionizing colostomy is useless.

SIGMOIDORECTAL INTUSSUSCEPTION

Although Allingham described this lesion in 1882 sigmoidorectal intussusception is a relatively uncommon clinical syndrome. Incarceration of this intussusception is rare. The sigmoidorectal intus-

susception is usually undiagnosed because the sigmoidoscopic examination is carried out with the patient in the knee chest position. This and the advancing sigmoidoscope generally reduce the intussusception so that a diagnosis is not made.

This type of obstruction depends for its formation upon the following factors: (1) abnormal mobility of the sigmoid due to redundancy with a long mesentery; (2) rigid fixation of the rectosigmoid and rectum by the sacral and pelvic fascia; and (3) a narrow caliber of the sigmoid colon as compared with the large lumen of the rectum. The weak anterior rectal wall is vulnerable to increased intra-abdominal pressure so that invagination of the narrow mobile sigmoid through the rectosigmoid and into the voluminous rectum occurs. Cramer and Pearl report two cases of incarceration of the intussusceptum in an obstruction of this type; however strangulation of an intussusceptum so incarcerated is extremely rare.

CHRONIC INTESTINAL OBSTRUCTION

One of the most difficult diagnostic problems facing the surgeon is the case with chronic intestinal obstruction. These patients are particularly difficult problems because even after the most meticulous clinical study the pathologic process may not be demonstrable. Most of these patients are diagnosed as neurasthenics, psychoneurotics, or unstable personality types and are shunted about for years until someone sufficiently interested and well informed finally makes the diagnosis. In such cases a normal gastro intestinal radiologic study performed in the usual fashion does not rule out chronic obstruction. The vast majority of these patients reported as normal by the routine radiologic technique may be diagnosed by means of special small bowel studies. The most important factor in the diagnosis of chronic intestinal obstruction is a strong suspicion on the part of the surgeon that he is dealing with a case of this type. Once such a suspicion has been aroused special small bowel studies coupled with a clinical history of the patient are sufficient for correct diagnosis. Although it is a matter of routine we utilize psychiatric consultation to evaluate the mental status of the patient; the final decision must remain with the surgeon. The two balloon Miller Abbott tube may be used to advantage in the radiologic study of this type of obstruction when the small bowel is involved. Using this tube selected segments of small bowel may be isolated and studied with a contrast medium. In addition pressure differences in various portions of the gastro intestinal tract may be measured. Pressure higher than that in the normal bowel may be expected in the presence of partial obstruction. In those cases where chronic obstruction is caused by a kinking together of loop

of bowel, the use of such tubes is of no value. In the final analysis a carefully taken history and a carefully performed examination especially during the intermittent attacks of obstruction are often sufficient to indicate the surgical approach. I cut this diagnostic Gordian knot.

Johnson studied the effect of partial small bowel obstruction in 1930. His studies suggested that chronic partial small bowel obstruction caused dilatation and functional disturbances of the more proximal small bowel in some fashion. Barber demonstrated in an experimental study in cats and dogs that partial obstruction of the ileum resulted in a dilatation of the duodenum. He pointed out that the relationship of the increased duodenal dilatability to distal obstructions was not completely the result of local disturbances. He believed that the duodenal dilatation was secondary and was due to an interference with the normal interlocking control of the neuromuscular mechanism. We have much to learn about the diagnosis and management of these chronically obstructed patients.

There are many diverse causes for chronic intestinal obstruction. Almost all portions of the gastro intestinal tract may be involved. The duodenum and small bowel are the sites of predilection.

CHRONIC OBSTRUCTION OF THE STOMACH

Pyloric stenosis is by far the most common cause of chronic obstruction of the stomach. Although an occasional case may be due to syphilitic fibrosis, in the vast majority of cases the stenosis is the result of fibrotic changes resulting from the healing of a pyloric ulcer. The patients continue

to prevent the clinical as well as the radiologic findings of chronic obstruction of the stomach for a long period of time. There is often considerable gastric back pressure with resultant gastric dilatation. Acute obstruction may supervene upon this chronically obstructed stomach and necessitate immediate hospitalization. When this occurs many of the patients may be treated by an indwelling Levin tube and continuous suction for three to four days during which time intravenous fluid are given. It is surprising how often the edema which is commonly the cause of the acute obstruction relents. When this occurs an elective subtotal gastrectomy may be performed as soon as the patient is in the optimum condition for surgery. The combination of local subincision of the inflammatory reaction plus a well prepared patient operated upon electively instead of in an emergency will materially lower the mortality and morbidity rates. However in those cases in which such conservative measures do not relieve the acute obstruction surgical intervention in the form of subtotal gastrectomy must be used whenever possible. In aged patients, poor risk patients, and those in whom subtotal gastrectomy with removal of the ulcer is technically dangerous a gastro-enterostomy with or without vagotomy may be the procedure of choice. Vagotomy should be performed in patients in the younger age group especially in those presenting high gastric acidities.

Gastric piles (prolapse of hypertrophic gastric mucosa through the pylorus) may produce intermittent attacks of obstruction at the gastric outlet. This type of chronic gastric obstruction is quite common. Although gastric piles are generally asymptomatic many cases of acute as well as chronic gastric obstruction have been reported as a result of the prolapse of this redundant gastric mucosa through the pylorus. A prolapse of this type usually reduces itself so that the obstruction becomes intermittent. Flaccid spasm around the prolapsed gastric fold may cause strangulation of this mucosa resulting in complete obstruction.

Pedunculated benign tumors or polyps of the stomach may produce chronic gastric obstruction by two mechanisms: (1) the tumor may produce a ball valve type of intermittent obstruction by resting against the pylorus or (2) the tumor may

pass through the pylorus obstructing the stomach and then may spontaneously reduce itself. This may recur for many years. Many of these patients who do not become acutely obstructed are chronically obstructed for years before proper surgical treatment is given.

CHRONIC DUODENAL OBSTRUCTION

Adhesions causing chronic duodenal obstruction may be congenital or acquired in origin. The cholecystoduodenocolonic band is a typical example of a congenital band which produces chronic duodenal obstruction. Inflammatory or post surgical adhesions resulting from disease of the liver, gall bladder, cystic duct or hepatic flexure of the colon may sufficiently compress the duodenum to chronically obstruct it.

Compression of the duodenum against the posterior abdominal wall with a resulting chronic duodenal obstruction may be caused by the pedicle of the superior mesenteric artery or by the middle colic artery. The theories advanced for this are presented in the chapter dealing with duodenal obstructions (Chapter 7).

Embryologic remnants persisting in the duodenum may cause chronic duodenal obstruction. The most common of these is the duodenal diaphragm. The persistence of such diaphragms into adult life is not uncommon. There is a great variation in the size of the lumen in these diaphragms and as a result the symptoms and back pressure in the stomach are inversely proportional to the size of this lumen. The smaller the opening in the diaphragm the more marked are the symptoms and the physical and radiologic findings. Chronic obstruction may occur over a period of years resulting in poor nutrition, retarded physical development and at times such mental disturbances as migraines. These chronic obstructions may become acute as a result of ingested foreign bodies such as fruit pits or coarse foods.

Cicatrizing duodenal ulcers are the most common cause for chronic duodenal obstruction. These are discussed in detail in the chapter on duodenal obstruction (Chapter 7).

The symptoms associated with duodenal obstruction are pain, nausea, vomiting and upper abdominal distention. Loss of weight is usually a

prominent feature of this disease. The pain usually comes on after meals, particularly large meals, and usually is located over the point of obstruction. Nausea and vomiting often follow the pain. If the site of the obstruction is distal to the ampulla of Vater, the vomitus contains bile. Following a seizure of vomiting, the pain and discomfort usually decrease and may disappear. The stomach only becomes dilated in those cases in which the chronic obstruction is fairly high grade and in which a long history is obtainable. Malnutrition and loss of weight are early and important findings.

A congenital short ligament of Treitz may cause intermittent duodenal obstruction by virtue of the sharp angulation that occurs between the fourth portion of the duodenum and the jejunum. At times the proximal jejunum may be weighted with intestinal contents, thus producing a sharp angulation at the duodenojejunal flexure which, being short and high, partially obstructs the duodenum at this point. Such partial obstructions are spontaneously relieved when the patient lies down. This type of partial obstruction becomes significant when paralytic ileus causes the jejunum to become so weighted with fluid that an acute obstruction occurs at the already partially obstructed duodenojejunal flexure.

CHRONIC SMALL BOWEL OBSTRUCTION

Adhesions are the cause of most of the chronic small bowel obstructions, although other etiologic factors have been reported. Chronic small bowel obstruction may be caused by any process that narrows its lumen sufficiently so that stasis of small bowel contents results. Some of the causes for this obstruction follow:

1. Kinking and angulation of the small bowel due to mesenteric cysts or tumors
2. Mesenteric inflammatory processes causing a degree of scarring sufficient to angulate and kink the bowel without completely obstructing it
3. Postoperative adhesions
4. Infections or abscess processes in the peritoneal cavity
5. Congenital adhesions
6. Matting syndrome

Kinking and angulation of the small bowel may be associated with space occupying lesions of the

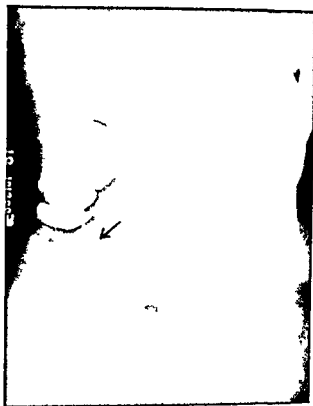


FIG. 209 Stenosis of the terminal ileum due to terminal ileitis



FIG. 210 Spot film of terminal ileum of patient in figure 209. Note ulcerative change.

me enteric. In addition, such me enteric tumors or cysts may become large enough to cause compression of the overlying small bowel with a resultant chronic obstruction. The signs and symptoms are generally vague. Cramping abdominal pain and intermittent abdominal distention are common. Radiologic study is invariably negative. The presence of an abdominal tumor mass which is movable, firm, and painless, associated with negative radiologic findings, should create a suspicion of a me enteric tumor. The cramping abdominal pain and intermittent distention would suggest partial small bowel obstruction associated with the mesenteric lesion.

Me enteric inflammatory processes such as abscesses, infected glands, and tuberculous me enteric glands may so scar the mesentery as to kink and partially obstruct the overlying bowel. These cases are extremely difficult to diagnose preoperatively. Small bowel study may demonstrate stasis of the intestinal stream in a dilated loop of bowel. This, in the absence of demonstrable narrowing of the bowel lumen, should suggest kinking as a causative factor. Chest X-ray and tuberculin tests may be helpful in tracking down the etiologic factor. In most instances, a correct preoperative diagnosis cannot be made. Many of these patients are treated for psychoneurosis.

Postoperative adhesions as a cause of chronic small bowel obstruction create a distressing problem for the surgeon. Many of these patients have been operated upon repeatedly only to find that the chronic obstruction becomes progressively worse with each exploratory laparotomy. This is understandable since each laparotomy increases the number of adhesions. All attempts to prevent the formation of these adhesions have failed. The only satisfactory methods for prevention of adhesions include the avoidance of unnecessary surgery, avoidance of hot packs, gentleness in handling tissues, absolute control of all bleeding points, and thorough peritonealization. Despite all these measures, those patients who have a propensity to fibroblastic proliferation tend to form extensive adhesions.

Congenital adhesions may cause recurrent or chronic intestinal obstruction. These adhesions develop from a disturbance in the embryologic proc-

esses and are not subject to human control. They are peculiarly difficult to diagnose. Diagnostically, it is helpful to keep the possibility of their presence in mind in any case in which chronic small bowel obstruction is suggested by the clinical history. A retroperitoneal position of the terminal ileum may result in chronic obstruction as a result of compression. This may be suggested by the radiologic demonstration of abnormal barium retention at this point.

The matting syndrome includes all those cases of chronic small bowel obstruction in which there is an extensive matting together of loops of small bowel and omentum. In such cases, the loops of bowel are matted to each other to such an extent that it precludes the possibility of dissecting them free. Chronic obstruction is almost invariably caused by the bowel so matted. Radiologic findings are generally negative. Many of these patients become drug addicts in an effort to relieve the attacks of pain. In those cases in which the matted bowel is not too extensive, primary resection of the entire matted bowel and end-to-end anastomosis of normal bowel is the procedure of choice. If the extent of the matting is such that resection is not possible, the Noble plication procedure may be useful.

Long-standing asymptomatic partial intestinal obstruction due to stricture of the ileum has been reported. As in the case of chronic duodenal obstruction, it is not uncommon for such obstruction to be made complete by the ingestion of some foreign body such as a fruit pit of large size. Burke, Mann, and Kirsh reported a case of this type in which a fruit pit remained in the ileum for 4 1/2 years and could not be excreted because of a stricture of the ileum at the site of a Meckel's diverticulum. The stenosis at the site of the diverticulum prevented the downward passage of the fruit pit. The diameter of the bowel at the site of stenosis was small enough to prevent the pit from wedging into it with the consequent development of acute intestinal obstruction, yet it was large enough to permit the passage of intestinal contents. The stenosis was never diagnosed prior to surgery, and the radiologic diagnosis in this case was regional ileitis.



FIG 211 Note the markedly narrowed point in the small bowel. This patient presented high grade partial intestinal obstruction.

CHRONIC OBSTRUCTION OF THE COLON

The types of chronic colonic obstruction are varied, ranging from functional disturbances to the organic stenosing lesions of the colon that cause partial obstruction. It has been suggested that these colonic obstructions produce symptoms not only because of their local effect of luminal narrowing but also because of a derangement of the entire gastrointestinal tract. Radiologic studies are difficult in these cases because the same individual may show evidence of stasis one day and at a later date the gastrointestinal study may be entirely normal.

In many patients with chronic colonic obstruction the history dates back to childhood. Repeated attacks of abdominal pain and distention in early childhood and early adult life are not uncommon. Constipation may increase in severity with the passage of time so that stronger cathartics are resorted to. Most of these people are diagnosed as psychoneurotics. They become apprehensive and

constantly seek medical attention which is usually ineffectual. The history is also complicated by the intermittent diarrhea so often associated with a chronically obstructed colon.

Foreign bodies, chronic granulomatous processes, and benign tumors may narrow the lumen of the colon to a degree sufficient to produce chronic intestinal obstruction. In these cases constipation is the most common symptom. This may be associated with a bloated sensation and a feeling of fullness although the bowels do move. Colicky pain occurring during these periods of abdominal distention is also suggestive of a partial obstruction. Generally, patients who suffer from chronic obstruction of the colon are a misunderstood and mistreated group of people. Radiologic demonstration of the obstructing mechanism is usually not obtainable except possibly by the use of the air contrast method. The markedly redundant colon so often reported as a variation of the normal may at times be responsible for chronic colonic obstruction and may produce symptoms that are distressing to the patient. It is possible that some of these can be improved by a resection of the redundant colon with end to end anastomosis. Although much needless surgery would doubtless be performed were all such patients treated in this fashion, there is little doubt that in well selected cases this operation can be curative. A case of this type was reported by Savignac where after many years of invalidism due to a chronically obstructed greatly redundant colon the patient was completely cured by resection of the redundant colon.

Among the developmental anomalies causing



FIG 212 Note marked abdominal distention associated with obstruction of the colon.

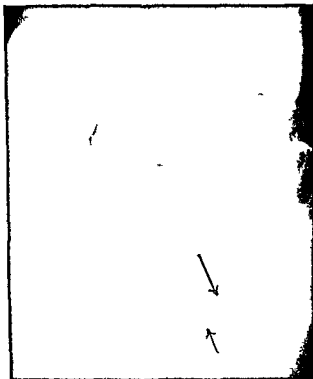


FIG. 213 Note marked compression of the junction of the descending colon and pelvic colon. This was due to compression of the colon against the bony pelvis by a retroperitoneal cyst.

chronic colonic obstruction are variations in the omental and peritoneal attachments which may cause segmental obstruction of the colon. Involution in the normal embryologic development of the omentum may result in omental folds pulling the hepatic flexure and the transverse colon together causing partial obstruction. A similar mechanism may partially obstruct the splenic flexure. These omental anomalies may consist of dense fibrous bands which cross, surround or even rotate segments of the colon.

The hepatic flexure of the colon or the splenic flexure may be retroperitoneally placed and as a result chronically obstructed. The fact that few such cases have been reported may be due in part to the extreme difficulty in diagnosing these lesions. The hepatic and splenic flexures are the most common sites for retroperitoneal placement of the colon, with the splenic flexure being involved most often. As much as 180 degrees of axial rotation of such retroperitoneal segments of bowel has been noted at operation. This is due to



FIG. 214 Transverse colon markedly dilated. Cyst held in forceps was opened and contents evacuated and then cyst removed.

fixation of one portion of the colon and torsion of the distal colon.

Chronic obstruction of the ascending colon may be the result of parietal colonic bands or membranes. Numerous case reports of this type have appeared and at one time numerous vague complaints were ascribed to this type of partial obstruction. In the early years of this century it was believed that the freeing of the ascending colon from these membranes would effectively relieve intestinal stasis. The initial wave of enthusiasm for this method was followed by a period of scepticism during which no surgery was believed indicated for lesions of this type. Now surgeons are again investigating the effect of such constricting bands and membranes with the result that some patients are being operated upon successfully. That these bands do occasionally produce chronic colonic obstruction has been demonstrated by the fact that some cases have been completely relieved after surgery.

The acquired type of bands producing partial colonic obstruction is often the result of cholecystitis, duodenal ulcer or resolution of a subphrenic abscess. When these bands are found in a patient presenting nonfixation of the right colon the chronic obstruction may be accompanied by intermittent attacks of complete obstruction due to cecal volvulus with the hepatic flexure as an axis. The intermittent colicky pain so often associated with chronic obstruction of the right colon is usually diagnosed as chronic appendicitis or chronic cholecystitis.

INTESTINAL OBSTRUCTION COMPLICATING OTHER MAJOR PATHOLOGY

Intestinal obstruction is at times associated with other major pathology. This association falls into three categories. First the obstructive process may be a direct result of the primary major pathology. Second the obstructive process may cause an existing pathologic process to take on major proportions. Third the obstructive process may not be related to the major pathology present in which case two serious pathologic processes are present at the same time.

The finding of an obstructive process anywhere along the gastro intestinal tract should not be accepted as the final and complete diagnosis until the physical findings, case history and laboratory studies prove that no other pathology is present. There is a tendency to diagnose intestinal obstruction and completely overlook other pathologic processes which may be present. This is particularly likely to occur with obstructive lesions of the sigmoid colon. Carcinoma at this site causing intestinal obstruction may at times be accompanied by a carcinoma of the right colon which is overlooked. Thus failure to conduct a complete study may result in a failure to treat the non obstructing carcinoma of the right colon. As a result two separate operative procedures would be required instead of one. Similarly an obstructing carcinoma at the hepatic flexure may be associated with a non obstructing carcinoma of the rectum. Here again because of an incomplete study the patient could be the victim of delay in treating the hidden lesion which lay all the while within the sigmoidoscopic range. Such errors can be avoided only by the realization that any patient with in-

testinal obstruction from whatever cause must be examined and treated as a whole and not as the victim of a single disease process. Sometimes intestinal obstruction is the first indication that another primary pathology is present.

Pyloric stenosis due to carcinoma of the stomach often makes its presence known by producing obstruction. In this case the pyloric stenosis and the carcinoma may be managed surgically at the same time since both require gastric resection and gastrojejunostomy. However when wide preterminal metastasis has occurred or in those instances in which a primary carcinoma of the pylorus has metastasized and obstructed the duodenum, the colon or other portions of the gastro intestinal tract the management of the primary lesion becomes secondary to that of the obstruction since the primary lesion cannot be cured. The only procedure indicated for such cases is palliation of the obstruction.

Syphilitic lesions of the stomach may be the only manifestation of the disease or may be associated with syphilitic lesions elsewhere in the body. In the latter event the lesions outside of the stomach are often far more significant than the gastric lesion.

Duodenal obstruction may occur because of compression of the duodenum by an aortic aneurysm. As in the case of duodenal obstruction caused by arterioenteric compression the correction of the duodenal obstruction cannot correct the etiologic factor. In cases of this sort diversionary gastrojejunostomy or duodenojejunostomy become the only surgical procedure possible.

Meckel's diverticulum is often associated with

serious heart disease which predisposes to its development. Emboli from the left ventricle formed there as a result of auricular fibrillation not infrequently cause mesenteric arterial occlusion with gangrene of the bowel. In this type of case the complication mesenteric occlusion far outweighs the primary disease process within the heart. Septic emboli from acute or subacute bacterial endocarditis may also cause mesenteric vascular occlusion. In most of these cases however the emboli cause small infarcts which are not incompatible with life whereas the endocarditis far outweighs the intestinal obstruction as a major pathologic process.

Obstruction of the sigmoid colon may accompany fractures of the left side of the pelvis. Such fractures are not uncommonly associated with bleeding around the sigmoid and descending colon. The scar tissue formed by the organization of such blood clots may so compress the colon as to obstruct it. Nelson reported a case of this type.

Retroperitoneal hemorrhage is almost invariably associated with paralytic ileus. Although the ileus is secondary to the primary pathology in the retroperitoneal area, Lennorant and Cordier emphasized the importance of the paralytic ileus as a complication in such cases. There has been considerable controversy as to the mechanism by which this paralytic ileus is produced. The irritant effect of retroperitoneal bleeding was demonstrated by Guilhal and Cuenot in an experimental study. It was believed that irritation of the splanchnic nervous system resulted in sympathetic stimulation causing paralytic ileus. Kuntz demonstrated that the retroperitoneal bleeding caused stimulation of both the sympathetic and the parasympathetic nervous systems. He showed that motor as well as inhibitory impulses to the gastrointestinal tract are conducted through the sympathetic and parasympathetic nervous systems with equal facility. The paralytic ileus in such cases develops from one to five days after the onset of the bleeding and is severe. The ileus does not respond to treatment until the stimulating bleeding has been stopped.

These retroperitoneal hemorrhages have been classified by Karabin as traumatic and nontraumatic. The traumatic group includes hemorrhages

resulting from direct injury to muscles, kidneys, blood vessels or other retroperitoneal tissue. Fractures of ribs, vertebrae or pelvis may be etiologic factors. Retroperitoneal bleeding as a result of surgical trauma upon such retroperitoneal structures as the retrocolonic appendix or kidney, rupture of vessels in broad ligaments or to the retroperitoneal vessels is also included in the traumatic group. Karabin places the following etiologic factors in the nontraumatic group:

1. Diseases of the retroperitoneal blood vessels.
2. Diseases of the blood such as leukemia, purpura, hemophilia.
3. Diseases of the kidney.
4. Diseases of the retroperitoneal tissues such as perinephritic infections or neoplasms.
5. Spontaneous retroperitoneal hemorrhage unassociated with any demonstrable pathologic etiologic process.

Lesions of the spinal cord may result in a paralytic ileus by cutting off the central motor nerve supply. Vertebral fractures with cord compression in the dorsal area result in immediate paralysis. This is commonly associated with considerable abdominal distention. Reduction of the fracture with release of cord compression often results in a return of peristaltic activity. Mediastinal tumors involving the vagus and sympathetic nerves also may result in a paralytic ileus. Hemorrhage, trauma or gunshot wounds to the mediastinum with injury to the vagus and sympathetic nerves invariably result in a marked degree of intestinal distention due to paralytic ileus. Diaphragmatic pleurisy is not infrequently associated with paralytic ileus due to splanchnic stimulation. In such cases treatment of the pleurisy brings a prompt subsidence of the ileus. Acute pancreatitis is commonly associated with some degree of paralytic ileus. The secondary intestinal distention may reach major proportions in such cases. As a result an erroneous diagnosis of mechanical small bowel obstruction is not uncommon.

Fecal impaction producing intestinal obstruction occurs fairly often in bedridden patients with multiple sclerosis, rheumatoid arthritis or any long standing degenerative process. The obstruction in all these cases is secondary to the primary incapacitating pathology.

The obstructive process may cause an existing pathologic condition to assume major proportions. As a good example of this we refer to the association of intestinal obstruction with hernia. In some of these cases the hernia although known to be present for many years may be asymptomatic. Incarceration of bowel within such hernial sacs immediately converts the asymptomatic pathology into a highly dangerous symptomatic one. Any type of hernia may become a surgical emergency if a portion of incarcerated or strangulated stomach, small bowel or colon becomes tripped within the hernial sac. Some minor trauma or increased intra-abdominal pressure may cause this to occur.

Diverticulosis may be present for years without making its presence known. The development of infection in the diverticulum converts this asymptomatic pathology into a symptomatic one by causing obstruction of the colon. In such cases the obstruction may take the form of paralytic ileus or mechanical obstruction due to compression of the colon by the inflammatory process.

Endometriosis may be present without symptoms. Involvement of the ileum or colon by the endometriosis however results in intestinal obstruction. This is a major pathologic process requiring immediate surgery. In cases of this type it is the complicating bowel obstruction that causes the endometriosis to take on major proportions.

Large gallstones may be found within the gall bladder in routine examinations without producing any symptoms. The slough of such large stones through the gall bladder wall into the duodenum immediately changes the benign innocuous gall bladder pathology into a major surgical condition with an associated high mortality rate. Intestinal obstruction produced by such large gallstones causes the existing pathology in the biliary tract to become of major importance.

In adult acute intestinal obstruction due to intussusception is not infrequently due to the presence of small polypoid benign asymptomatic tumors. Such tumors in themselves are innocuous but when intussusception is produced by their presence within the bowel lumen they immediately take on major surgical proportions.

Intestinal obstruction may occur as an incidental

finding in a patient suffering from other major pathology. In such cases although the intestinal obstruction is important the associated major pathology may be of even greater importance. Insofar as the life of the patient is concerned in these cases the obstruction occurs independently and is unassociated etiologically with the other major pathology.

Prepyloric ulcers causing gastric obstruction may be found in a patient who has a non-obstructive and asymptomatic carcinoma of the rectum. Bleeding posterior wall duodenal ulcers followed by stenosis and obstruction of the duodenum may be found in the same patient with colonic neoplasms. In such cases the rectal bleeding may be ascribed to the obvious duodenal lesion and no attempt made to study the gastro-intestinal tract in its entirety. Thus obvious lesions may mask and hide secondary pathologic conditions of greater consequence.

Intestinal obstruction caused by incarcerated or strangulated hernia may be associated with major disease in the heart, kidneys, liver or pancreas. The major pathology may be due to carcinoma of the colon, kidney, prostate or female generative organs. As a result of straining at stool or increased intra-abdominal pressure incarceration of pre-existing hernia may occur. In such cases the obstruction must be treated before all other pathology as the major pathologic condition present. It demands immediate surgical treatment regardless of the condition of the patient unless of course such surgical intervention would be fatal. In cases of this type the least traumatic procedure to relieve the obstruction should be used. This may consist of simply incising the hernial sac to create a small bowel fistula. Once the acute obstruction has been corrected further definitive treatment may be instituted at a later time when the condition of the patient is less precarious. However in some patients for instance one deeply jaundiced due to carcinoma of the head of the pancreas and obstructed because of a strangulated hernia both conditions require urgent treatment. The correction of the strangulated hernia must be performed first and then shortly thereafter the obstructive jaundice should be treated.

Obstructions in the gastrointestinal tract particularly in the older age groups are not infrequently associated with major cardiac pathology. Valvular heart disease, coronary disease and myocardial degenerative changes of severe grade are not infrequently found in the obstructed patient. In such cases the surgeon must decide whether the risk of surgery outweighs the risk to the patient of not correcting the obstruction. Since a decision of this type can be difficult, the surgeon through fear of the cardiac status may often be swayed into the use of conservative measures long beyond the time which judgment indicates to be desirable for surgery. In these cases the co treatment with an internist is helpful in evaluating the risk potential.

OBSTRUCTION IN INFANCY AND CHILDHOOD

CLIFFORD D. BLISSON, M.D.

The management of intestinal obstruction in infants and children differs considerably from that in adults. It is extremely dangerous to take the attitude that infants and children are miniature adults. On the contrary, from the point of view of treatment for intestinal obstruction they must be considered as different organisms having different physiologic mechanisms and requiring different methods of diagnosis and treatment. An early diagnosis of acute intestinal obstruction in infancy and childhood has become increasingly important since over the past 10 years the use of antibiotics plus better surgical technics based upon our improved understanding of the physiology and body chemistry have greatly increased the possibility of cure.

TECHNIC OF EXAMINATION

Survival of infants with complete intestinal obstruction depends upon early diagnosis and early operative treatment. A carefully taken history is of great importance and sufficient time should be allowed so that an accurate history may be obtained. It is essential that one consider the possibility of exanthema or respiratory diseases as well as genito-urinary disorders. All of these are capable of producing abdominal symptoms in these small patient.

The examination of the infant should be carried out in an unhurried fashion and every effort made to win the confidence of the child so that he will be relaxed. Because the younger patients are un-

able to cooperate with the examining physician considerable emphasis must be placed upon a careful physical examination in order to arrive at a correct diagnosis. Although the exact nature of the obstructing lesion sometimes can not be determined except at laparotomy, it is usually possible to arrive at a reasonably exact diagnosis prior to surgery. In general infants suspected of having an intestinal obstruction fall into three main groups. The first group is composed of those children in whom the diagnosis is obvious. In this group one would find incarcerated or strangulated hernias, ruptured omphaloceles, and such lesions as pyloric stenosis, all of which lend themselves to diagnostic study. In these patients particularly those presenting incarcerated hernias can an intestinal obstruction the diagnosis can be made with absolute certainty. In the second group are those infants for whom a reasonably accurate diagnosis not only of the intestinal obstruction but also its etiology is possible. In this group one might find intussusception, Hirschsprung's disease, duodenal atresia, and in the newborn some congenital anomaly as the source of the obstruction. The third group consists of those infant or children in whom an accurate diagnosis prior to surgery is impossible. Here the belief that one can hope for a diagnosis that some intestinal obstruction is present requiring operative intervention. In this group one finds obstruction is a result of Meckel's diverticulum, congenital band, duplica-

tions of the gastro intestinal tract with obstruction meconium ileus and volvulus

A systematic examination should be carried out beginning with the mouth ears and throat being constantly on the alert for such signs of examination as Koplick's spots. Any diagnostic procedure which might be painful or which may cause discomfort to the child should be deferred until the abdominal examination has been made

Points to look for in the examination of the abdomen are the presence and degree of abdominal distention and the presence or absence of peristaltic waves. First auscultation of the abdomen should be performed to determine the presence and degree of peristaltic activity. Hyperperistalsis and borborygmus particularly when associated with attacks of cramping pain should be looked for. The absence of peristaltic activity is as significant as its presence. Following auscultation the abdomen should be palpated gently beginning in the quadrant farthest away from the area suspected to contain the obstructive lesion. An attempt should be made to divert the child's attention while carrying on this palpation. The entire abdomen should be palpated systematically two or three times each time more deeply than before. In this way localized points of tenderness muscle spasm or intra abdominal masses may be noted. It may be difficult at times to distinguish between voluntary and involuntary spasm of the abdominal muscles. In order to obtain relaxation of the abdominal musculature the child should be given sufficient time to relax. In some cases a mild sedative may be given to secure this relaxation. With proper relaxation an intra abdominal mass may be palpable which otherwise might not have been felt. Once the examination has been concluded a rectal examination should be made. The palpation of a rectal mass the presence or absence of stool in the rectal ampulla and the presence of blood in the stool are significant findings.

Laboratory studies can be extremely helpful in two ways. First they may yield information which aids in arriving at a correct diagnosis and second laboratory data may be of value in making a decision as to the surgical risk involved in instituting treatment. The degree of dehydration alkalosis or acidosis and the presence or absence of anemia are

of value in determining prognosis. At this point one should bear in mind the fact that the hemoglobin in the newborn infant is normally higher than average while it tends normally to be low between the age of two months and two years. In the very small infant the white blood count may be normal or depressed even in the presence of a massive infection. Furthermore it must be remembered that lymphocytosis is a normal condition for children under the age of five. In such patients a rise of polymorphonuclear cells to 60 per cent of the total white count represents a leukocytosis.

Following the physical examination a survey film of the abdomen should be made. Considerable care must be exercised in the interpretation of the X ray in infants and small children since intestinal gas is normally present in the small bowel of such infants. The amount of gas diminishes rapidly after the age of two so that in children five years old little gas is found in the gastro intestinal tract.

GENERAL MANAGEMENT OF INTESTINAL OBSTRUCTION IN CHILDREN

It has been repeatedly emphasized that an infant presents a far better surgical risk during the first 48 hours after birth than it does a week later. This is particularly the case in the management of congenital high obstructions. Because of the rapid deterioration of the infant with the passage of time surgical intervention should be undertaken as soon as possible after birth and therefore a correct diagnosis at the earliest possible moment is imperative. Without surgery death invariably occurs within a week when the obstruction is complete.

Infants and children do not tolerate the loss of fluids as well as adults and in addition nutritional deficiencies are far more serious. For this reason adequate hydration and feeding at the earliest possible moment are of the greatest importance in the management of children with intestinal obstruction. Adynamic ileus is a particularly serious complication in children. Individuals in this age group react to distention of the intestinal tract far less well than do adults. Intestinal intubation is both practical and easily accomplished in children.

The pre operative and postoperative care of the infant is important. The intake of fluid should be maintained at 3 ounces per pound of body weight for a 24 hour period. This may be increased to compensate for loss of body fluids through vomiting or the use of a gastric tube. Intravenous alimentation should be used postoperatively as long as gastric suction is being maintained. Great care should be taken to avoid overhydration. Oral feedings should be withheld in the early postoperative period during the time that gastric suction is being used. In the presence of shock or loss of protein blood may be given intravenously. The feeding problem is an extremely difficult one and should be maintained under the close supervision of a pediatrician. The diet of these infants should be supplemented with the essential amounts of vitamins. In the newborn and the premature oxygen inhalation by means of an oxygen tent is desirable. Proper antibiotics should be given to control and combat infection. A properly given diet should be instituted as soon as feasible. This may be begun at times with a Levin tube in place. The tube should be clamped off for short periods of time to permit the ingested food from the stomach to pass through the gastrointestinal tract. Diarrhea and intestinal upsets may occur as a result of improperly given food so that the loss of water through diarrhea is well as suction may result in a serious dehydration problem. Because most newborn infants have a low concentration of prothrombin in the blood the administration of vitamin K has been suggested for all cases of obstruction in newborn infants. Premature infants and newborns particularly those under 6 pounds are best taken care of in a well ventilated incubator.

ETIOLOGY AND MANAGEMENT OF OBSTRUCTION IN INFANCY AND CHILDHOOD

Intestinal obstruction in infancy and childhood may be classified in two well defined groups. In the first group we find all those patients in whom the etiology of the obstruction is purely congenital in origin and in the second group are found all those patients in whom the obstruction is postnatal. Although many of the obstructions in this latter group are definitely acquired in the sense that

there is no predisposing congenital factor yet in some instances such obstruction occurs long after birth as a result of a developmental anomaly. There is then of necessity some overlapping of the congenital and postnatal etiologic mechanisms of obstruction in this second group.

Congenital

There are many congenital causes for obstruction of the alimentary tract in infancy and childhood. Among these congenital anomalies are atresia of the esophagus, intrinsic obstruction of the stomach, pyloric stenosis, intrinsic obstruction of the duodenum, duodenal diaphragm, atresia or stenosis of the small bowel, duplications of the small bowel, mesenteric cysts, aganglion of the mesenteric plexus producing congenital megacolon and anomalies of the colon and rectum. In addition to these one must consider anomalies of intestinal rotation which may result in intestinal obstruction by several mechanisms the most important of which is volvulus.

Esophageal Atresia

The incidence of esophageal atresia has not been definitely established. It was thought to be an extremely rare lesion. Many authors report having seen this lesion only once in 50,000 newborn. Haught believes that an incidence of 1 in 2000 births is more nearly correct.

Types. Congenital esophageal atresia has been divided into three main groups by Haught:

1. Those patients in whom the esophagus is absent or in whom it is merely a cord.
2. Cases in which there is an upper and lower segment each ending in a blind pouch.
3. Those infants with fistulous communication between the esophageal segment and the trachea or bronchus.

Diagnosis. Esophageal obstruction should be suspected in any infant who begins to vomit immediately after birth and has choking, dyspnea and cyanosis associated with the vomiting. Further study may be carried out by passing a small rubber catheter into the esophagus. If the pressure of the catheter is obstructed about 10 cm from the lower jaw 1 to 2 cc of hypodermic may be injected into the catheter and an x-ray study made.

These studies will usually demonstrate the esophageal segment terminating at the level of the second or third dorsal vertebra. By means of careful radiographic examination the true diagnosis of the type of anomaly present may be made. It is essential in such cases that treatment be given at the earliest possible time.

Treatment A discussion of the treatment of the various types of congenital atresia of the esophagus is presented in Chapter 5.

Intrinsic Obstruction of the Stomach

Intrinsic obstruction of the stomach in infancy is a rather rarely found lesion. Touroff reported the first successful operation for this anomaly in 1940. A second patient successfully operated upon for intrinsic obstruction of the stomach was reported by Benson and Coury in 1951. In both patients the stomach was completely obstructed by a prepyloric diaphragm. This author has noted that these patients were often prematures. He has also noted that other associated congenital anomalies were factors in the high mortality rate associated with this congenital anomaly in the past.

Diagnosis These infants often premature begin to vomit immediately after birth. They are unable to retain either water or milk. Bile is absent from the vomitus. A diagnosis of obstruction at the outlet of the stomach may be readily made by the insertion of a small caliber Levin tube and the injection of a small amount of lipiodol or dilute barium. If air is used as a contrast medium it may be injected via the Levin tube. An accurate diagnosis may be made by using any of these media.

Treatment The patient successfully operated upon by Touroff was treated by means of multiple incisions of the membrane and by pyloroplasty. The patient reported by Benson and Coury was treated successfully by excision of the diaphragm.

Obstruction of the Duodenum

These obstructions are relatively common when compared to intrinsic gastric obstructions. The first case of duodenal atresia described by Calder in 1733 was an autopsy finding. The first successful operation for complete intrinsic duodenal obstruction was reported by Ernst in 1916. From 1916 until 1952 according to a review of the liter-

ature by the author there were only 57 successfully treated infants with duodenal atresia or stenosis. In a review of 400 infants admitted to the Children's Hospital in Michigan with a diagnosis of intestinal obstruction over a 7 year period 16 per cent were found to have atresia of some portion of the gastro intestinal tract and 2 per cent of these patients had obstruction of the duodenum.

Types The factors producing duodenal obstruction may be classified into two main groups: (1) intrinsic or acting within the duodenum and (2) extrinsic as a result of compression from outside.

The intrinsic factor usually has its beginning during the formation of the intestinal tract and begins between the fifth and tenth weeks of fetal life. At this time the intestine changes from a solid core to its final form with a lumen lined by a well defined epithelium. A failure of vacuolization at the time the lumen is formed in the duodenum is responsible for congenital obstructions of the duodenum. A failure of complete vacuolization may result in an actual solid core of cells obstructing the duodenum. If the vacuolization is partial various degrees of membrane formation permitting incomplete obstruction of the duodenum may occur. As a result there may be varying degrees of duodenal obstruction or there may be a combination of complete obstruction with areas of partial obstruction.

The gross appearance of the bowel in the region of the duodenal diaphragm is apt to be normal to inspection and palpation. Some degree of dilatation proximal to the point of obstruction is almost always found. This depends upon the degree of obstruction and its duration. In many cases the obstructive diaphragm is a thin partition extending transversely across the lumen of the bowel. It may be complete or it may contain an aperture of variable size. There is a very definite relationship in the size of the aperture of the diaphragm to the age of the patient and the time of onset of symptoms. The size of the aperture in the diaphragm does not increase in proportion to the normal lumen of the bowel and therefore symptoms appear when the disproportion becomes favorable for obstruction.

It is important to remember that multiple sites

of atresia may occur along multiple points in the duodenum. In 1845 Robert Boyd reported the first case of obstruction of the duodenum due to a double diaphragm. Since then numerous cases have been reported and it has been estimated that duodenal diaphragm occurs in 2 per cent of all obstructions. In the series of cases reported by Benson and Coury, one patient had two separate intrinsic duodenal obstructions; one was a mongoloid and a third had a congenital cardiac anomaly. Bodian, White, Carter, and Low report a high incidence of mongolism in infants suffering from intrinsic malformations of the duodenum. They report an overall frequency of approximately one in every three duodenal obstructions. As a result of their experience during 1951 in which five consecutive cases of duodenal atresia or stenosis were found to be mongoloids, they suggest that the frequency of mongolism may be even higher than one in three cases. Their explanation for the paucity of recorded cases is the fact that mongoloids generally do not come under medical observation until the end of infancy or even later when there is obvious retardation of mental development. Most cases of duodenal atresia or stenosis generally die long before this time and the condition of mongolism would therefore go undiagnosed. These authors emphasize the fact that no single feature is peculiar to mongolism nor is any sign of invariable occurrence, but the presence of a cherry red spot around the macula is almost pathognomonic of Mongolian idiocy before there is other evidence of mental retardation.

In a case reported by Morton, the duodenal diaphragm was complicated by an incarcerated para-duodenal fossa hernia near the ligament of Treitz.

The chief causes of extrinsic obstruction of the duodenum are generally found in the second or third limbs of the duodenum and are normally the result of incomplete rotation of the cecum and colon causing partial or complete obstruction of the second or third duodenal limbs. Congenital bands which produce angulation or actual compression of the duodenum are another cause of extrinsic duodenal obstruction.

Incomplete rotation of the cecum during the first 6 to 10 weeks of fetal life predisposes to the development of extrinsic obstruction of the second



FIG. 215 Note the extensive congenital band producing duodenal compression.

and the third limb of the duodenum. In such case the obstruction of the duodenum may be complete at the time of birth or may not become complete until several weeks or months later. There are many cases in which duodenal obstruction from this cause never becomes complete.

One of the most unusual combinations of anomaly with obstruction of the duodenum is the case report of Fox and Crawford. In this case a 14-month-old infant presented a duodenal obstruction caused by congenital bands with situs viscerum versus transposition of all organs, nonrotation of the colon, and an absent spleen.

Age. Duodenal obstruction may be encountered at any time in life. The youngest patient reported was a stillbirth and the oldest was a patient 70 years of age. However, in 38 per cent of all cases reported, the symptoms of obstruction began during the first two weeks of life.

Diagnosis. The presence of a complete obstruction of the duodenum, regardless of its etiology, manifests itself a day or two after birth. Vomiting

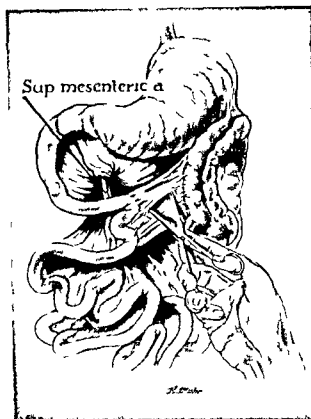


FIG 216 All these congenital bands must be carefully cut if good results are to be obtained

ing is an early symptom. In many cases this may be the main symptom. The vomiting in duodenal obstruction differs from that noted in pyloric stenosis in that it occurs on the first or second day of life rather than one to three weeks after birth as is the case in pyloric stenosis. Moreover the vomiting of duodenal obstruction is not the projectile type seen in pyloric stenosis. In addition in those cases in which the obstruction of the duodenum is distal to the ampulla of Vater the vomitus will contain bile which is not true of the vomitus in pyloric stenosis. When the obstruction is incomplete the child may be treated for many weeks or months as a feeding problem and a correct diagnosis generally is not made until some time later. In such cases the symptoms of obstruction may not become obvious for days, weeks, months or years depending upon the degree of duodenal obstruction.

When the obstruction involves the third limb of the duodenum examination may disclose a rather



FIG 217 Cecum and ascending colon were found in an abnormal anatomic position

marked distention limited to the upper abdomen. In those infants with incomplete duodenal obstruction in which an incorrect diagnosis of a behavior or feeding problem has been made medical treatment almost invariably produces little or no improvement. Although the obstruction is of a relatively high grade there may be sufficient food and liquids seeping through to keep the infant at the lowest subsistence level. These children are always undernourished and underdeveloped. They show marked muscular and skeletal retardation. Generally at the time the diagnosis is made they appear much younger than their chronologic age. It is surprising how rapidly these infants regain their normal growth and development following correction of the duodenal obstruction.

In complete obstruction of the duodenum a survey film of the abdomen shows that the gas is limited to the stomach and duodenum with no gas found distal to the point of obstruction. In those cases of duodenal stenosis in which the obstruction is incomplete gas may be found not only in the

stomach and duodenum but also in the small bowel and colon. In such cases dilute barium may be given by mouth and X rays taken in order to localize the point of obstruction. In general barium should not be given by mouth in the diagnosis of duodenal obstruction because of the danger of the infant aspirating the vomited barium. As the characteristic findings of high grade or complete duodenal obstruction, radiologic studies demonstrate a markedly dilated and gas filled stomach and duodenum with little or no gas in the small bowel.

Treatment Obstruction of the duodenum in infancy requires surgery at the earliest possible moment. This is essential before dehydration and electrolyte imbalance appear to a degree that endangers the life of the patient.

In the surgical management of the duodenal obstruction a diversionary procedure is essential if the child is to survive. The procedure of choice for obstructions at or below the ampulla of Vater is a gastro enterostomy or a duodenojejunostomy. Duodenojejunostomy is preferred by most surgeons because it drains the duodenum better and tends to prevent neutralization of the gastric secretions. Figure 218 demonstrates anterior gastro enterostomy for those cases in which the obstruction is in the first portion of the duodenum and above the ampulla of Vater. Figure 219 is an antecolic duodenojejunostomy for obstructions below the ampulla of Vater. Figure 220 is a transmesocolic type of duodenojejunostomy useful in selected cases.

In those cases of obstruction of the duodenum due to a diaphragm at the time of surgery the obstruction often cannot be detected without opening the duodenum. The only clue to the presence of an obstructive lesion of this type may be a dilatation of the stomach and duodenum proximal to the point of obstruction. When the obstruction has been relieved either by a diversionary surgical procedure or if the condition of the patient permits by incising the duodenal diaphragm a careful search should be made in the remainder of the gastrointestinal tract for other anomalies in development. Minor anomalies in development which will not jeopardize the survival of the patient



FIG 218 Gastrojejunostomy for obstruction of the duodenum. This is a simple procedure and gives good results.

should not be treated at the time of surgery for duodenal obstruction. Their presence should be noted however for further reference.

When congenital bands produce an extrinsic duodenal obstruction, division of all of the bands causing duodenal compression is the required surgical treatment. This permits the colon to rest on the left side of the abdomen and frees the duodenum of all compression. No attempt should be made to place and fix the cecum and the ascending colon in their normal anatomic positions. In an occasional case there may be incomplete rotation of the cecum associated with incomplete fixation of the mesentery of the terminal jejunum and ileum. As a result a segmental small bowel volvulus may occur. The superior mesenteric artery furnishes the axis of rotation in such cases. Detorsion of the volvulus is sufficient to correct the type of obstruction.

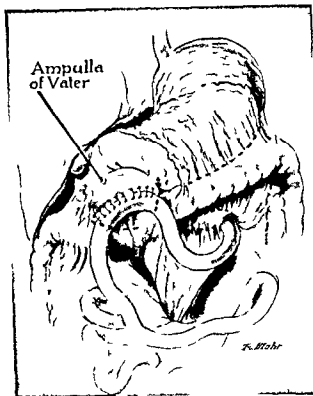


FIG 219 Duodenojejunostomy for obstruction below the ampulla of Vater

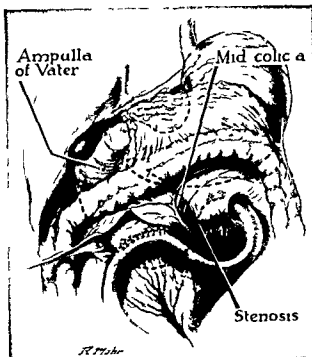


FIG 220 Transmesocolomic duodenojejunostomy This operation is desirable whenever possible for the management of infra ampullary duodenal obstructions

Congenital Hypertrophic Stenosis of the Pylorus

Congenital hypertrophic stenosis of the pylorus is the most common obstructive condition of infancy and usually requires surgical treatment in the first two months of life. It has been reported as occurring once in every 500 births. Although the etiology of this condition has not been established many pediatricians believe that it is due to a pre-existing pylorospasm which produces a hypertrophy of the pyloric muscles. Pathologically there is a hyperplasia and hypertrophy of the pyloric sphincter muscle producing varying degrees of obstruction.

The incidence of pyloric stenosis in twins was studied by Metrakos. In a study of four pairs of monozygotic twins both twins were found to be affected in three of the pairs and one twin was affected in the fourth pair. In one pair of dizygotic twins only one twin was affected. These five pairs of twins are combined with 127 pairs reported in the literature and an analysis was made to determine the hereditary tendency toward pyloric ste-

nosis. This study revealed that there could be little doubt that a hereditary factor was involved in the etiology of hypertrophic pyloric stenosis. It also appeared that some prenatal or postnatal factor might be necessary for the development of the hypertrophy and that such an environmental factor might be effective in only one of a pair of twins even when the twins were genetically alike.

Diagnosis Symptoms of pyloric stenosis occur between the second and fifth weeks of life. Persistent vomiting of the projectile type is a very characteristic symptom. The vomitus contains recently ingested food and gastric contents but no bile. In many cases repeated and careful examinations of the abdomen will demonstrate the presence of an abdominal spindle shaped tumor mass in the region of the pylorus. This examination should be made with the child in the prone position which permits the stomach to fall forward. Once a definite pyloric mass is palpable a diagnosis of hypertrophic pyloric stenosis is almost certain. Although inability to palpate a pyloric mass is not definite evidence that hypertrophic pyloric stenosis is not present it should however lead one to

suspect the possibility of atresia or stenosis of the duodenum particularly in those cases in which some bile is found in the vomitus

The administration of a thin barium meal may be required in order to establish a correct diagnosis. Carefully made radiographs will invariably demonstrate a string sign indicating the narrow pyloric canal through which the barium passes. Since it is not at all uncommon for this diagnosis to be missed by the inexperienced radiologist it is essential that competent and experienced radiologists make such studies.

Treatment. Hypertrophic pyloric stenosis never constitutes a surgical emergency. Infants admitted for the treatment of hypertrophic pyloric stenosis are usually dehydrated and in poor electrolyte balance. In such cases sufficient time should be taken to correct the dehydration and the electrolyte imbalance before subjecting the infant to surgery. One two or three days may be required to accomplish this. In addition to correcting these imbalances it may be necessary to give a small blood transfusion in order to bring the plasma protein level as close to normal as possible.

In 1908 Fredet advocated splitting the hypertrophied muscle in a longitudinal fashion and resuturing it in a transverse direction. In 1912 Kamstedt described the operation which is now universally accepted. This operation consists of splitting the hypertrophied muscle longitudinally but not incising the mucosa thus permitting the mucosa to bulge between the muscle fibers. This increases the lumen of the duodenum. No attempt is made to suture the muscle. As a result of this surgical procedure the mortality rate associated with the treatment of pyloric stenosis is now in the neighborhood of 1 per cent. Reviewing a series of infants from the surgical service at the Children's Hospital of Michigan and from my private service at Harper Hospital I have found 517 infants operated upon for pyloric stenosis with four deaths or a mortality rate of 0.77 per cent. In this group of 517 infants 425 were males and 92 females.

Atresia of the Small Intestine

Atresia is derived from the Greek word *atrea* meaning nonperforated. This term is used in medical literature to describe the absence or

closure of a lumen or channel in the small bowel or in any hollow viscus.

It has been estimated that intrinsic defects of the small bowel occur once in every 20 000 births. Although there are numerous theories to explain these defects their exact etiology is unknown. Atresia may occur at any level of the gastrointestinal tract but is most frequent in the terminal ileum. The obstruction may vary from a mild constriction due to the presence of a diaphragm to a complete interruption of the continuity of the gastrointestinal tract with separation of the blind end and with an associated defect in the mesentery. In approximately 15 per cent of the cases multiple strictures producing more than one point of obstruction are found. The strictures may be either continuous or widely separated. One of the first reports of a case of total atresia of the terminal ileum was made by Gocler in 1683.

The occurrence of an identical type of intestinal obstruction in identical twins appears to be extremely rare judging from the paucity of material available on this subject. Lewis reports one instance of complete intestinal obstruction in identical twins due to exactly the same mechanism. The cause of the obstruction in each case was a membrane of the ileocecal valve which completely closed the bowel at this point.

Diagnosis. The inability of infants and children to withstand distention and acute fluid losses make early recognition and immediate definitive treatment essential. Ladd's dictum that "Any child who continues to vomit during the first day or two of life while being fed a normal amount of breast milk or a reasonable formula should be investigated radiographically for intestinal obstruction" should be very seriously considered by all surgeons who undertake the treatment of children.

The clinical picture in intestinal atresia varies with the degree of small bowel obstruction and the level along the small bowel that the obstruction is found. Generally the symptoms begin soon after birth. In high intestinal obstruction like obstruction of the jejunum symptoms occur with the first feedings. Vomitus containing bile is one of the earliest symptoms noted. However in low intestinal obstruction intestinal distention is a prom-

ment feature in such cases the vomiting appears several days later.

Farber has proposed an interesting laboratory method for early diagnosis of congenital atresia of the alimentary tract. This procedure is based upon a constant presence of cornified epithelial cells in the normal meconium. These cells are derived from the skin of the fetus which is swallowed with the other amniotic sac contents to contribute to the formation of the meconium. A microscopic examination of smears of normal meconium when treated with ether stained with Sterling's gentian violet and decolorized by acid alcohol reveals large numbers of cornified epithelial cells. Since all the other cells are decolorized by this method the cornified cells are easily recognizable. The absence of cornified epithelial cells in smears of meconium is considered by Farber as proof of the existence of congenital atresia of the alimentary tract.

A carefully taken history and a painstaking physical examination utilizing radiologic study and the Farber's laboratory test permit an accurate diagnosis in most cases of atresia of the small bowel. However, in those patients in whom the obstruction is not complete but is a high grade partial variety, a correct diagnosis may be extremely difficult. These patients with partial obstruction may present diarrhea as the most prominent feature of the disease.

Treatment. The methods of treatment used to correct stenosis or atresia of the small bowel are divided into three large groups:

1. **Enterostomy.**—Normally, this treatment meets with failure because of the inability of the infant to keep up with the marked fluid and electrolyte losses as well as the nutritional deficiencies.
2. **Double-barrelled enterostomy.**—This has been successful in a few cases. However, the difficulties in maintaining adequate nutrition and keeping the infant in water and electrolyte balance are such that it has very little usefulness except as a temporary measure.
3. **Resection decompression of proximal loop and primary anastomosis.**—either end to end or side to side with resection of the obstructing lesion.—This is the procedure of

choice. This method has been shown to give the best end results. In recent years the mortality rate associated with this procedure has been reduced considerably.

The success attained in the management of intestinal atresia depends upon the speed with which a correct diagnosis has been made and the rapidity with which surgical treatment is instituted. In those cases in which early diagnosis has not been made, the obstruction goes on to perforation and peritonitis. At the present time it is realized that except in those instances in which the condition of the patient is prohibitive, the procedure of choice is primary resection and anastomosis.

The method we have found to yield the best results in the treatment of atresia of the jejunum and ileum is:

1. Resection of the bulbous blind end of the proximal loop.
2. Decompression of the proximal segment of gas and fluid.
3. Primary anastomosis. This may be side to side, end to end, or end to side depending upon the segment of small intestine resected. For those atresias near the terminal ileum an end to side ileocolostomy is most satisfactory.

Baronofsky, writing of primary resection and anastomosis in obstruction of the small bowel due to atresia, mentions the use of a single row of fine silk Lembert sutures. In those instances in which there is a marked disproportion between the two loops of bowel to be anastomosed, the loop of bowel with the smaller lumen may be anastomosed obliquely and an end-to-end anastomosis performed. It has been demonstrated experimentally that the lumen so obtained is generally adequate and functional in nature.

Atresia of the Colon

Congenital diaphragm or atresia is an uncommon cause of obstruction in the colon. The first case with total atresia of the colon was reported by Bimlinger in 1673. Intestinal stenosis when it involves the colon may consist only of a low grade narrowing of the passageway which is not incompatible with life and which may not be discovered

for many years. At times a narrow ringlike or diaphragm like constriction may be noted in the colon and it may affect segments of bowel several inches long. Occasionally atresias and stenosis of the colon are multiple appearing at more than one level. W. A. Evans in reviewing the incidence of this disorder has estimated that there is one case of atresia of the colon or stenosis in every 1500 births.

Diagnosis. Any infant who presents signs of intestinal obstruction days after birth and who has had no meconium stools in the presence of a patent anus is quite possibly the victim of a colonic obstruction. Because obstructions in the colon are far down in the gastro intestinal tract vomiting occurs as a late symptom. Fever and abdominal distention may precede the vomiting by days. In many cases in which the sigmoid colon is obstructed in infancy fever may be a very prominent feature of the disease. This is either the result of dehydration or of a perforation of the distended bowel with subsequent peritonitis.

Although atresia of the ascending colon is uncommon having been rarely reported, Jemson reported a case which was successfully treated surgically.

In examining an X-ray survey film taken shortly after birth it is sometimes difficult to differentiate between the small bowel and the colon. When the entire gastro intestinal tract including the colon is seen to be distended with gas the obstruction is obviously low in the colon. In such cases atresia of the colon must be considered and looked for. The presence of a microcolon is not indicative of a primary congenital anomaly when found at operation. It is however indicative of the fact that the colon has never been filled because of the obstruction in the proximal bowel. The pencil sized colon is intrinsically normal. In such a case if the fecal stream can be brought into the colon it will dilate and undergo enlargement until a normal size is reached.

Treatment. There are relatively few cases of congenital atresia of the colon which have been successfully treated reported in the surgical literature to date. The surgical management of obstruction of the colon varies depending upon whether the point of obstruction is in the right or left

colon. In general primary resection and anastomosis are considered to be the treatment of choice for colonic atresic lesions. Many surgeons prefer a side to side anastomosis although the trend is definitely toward an end to end anastomosis in one or two layers. In all of the patients taken to surgery a rectal tube should be placed well up into the rectum or sigmoid if possible.

Decompression of the small bowel in the newborn is rarely possible by means of the long intestinal decompression tube in obstructions of the colon. The routine use of the Levin tube however is of value in keeping the stomach empty and free of gas and aspirated air. The abdominal incision should be of sufficient length to permit accurate intra abdominal exploration. It must be constantly borne in mind that such infants may present multiple anomalies. These should be looked for. It is especially important that any possible small bowel obstruction associated with the atresia of the colon be found. For this reason before the colonic lesion is treated surgically the intra abdominal examination should begin with the stomach and systematically include the entire gastro intestinal tract. Enterotomy and exteriorization procedures should be avoided if possible in infant surgery. However if there is much distention of the proximal loop an exteriorizing procedure is preferable and definitive anastomosis may be postponed until a later date. Generally it is desirable to reestablish the intestinal continuity at the time of surgery in order to reduce fluid loss.

Malformations of the Rectum and Anus

Malformations of the anus and rectum are said to occur about once in every 1000 births. They may occur either as an atresia or a stenosis of the rectum alone or in conjunction with fistulous communication with the urogenital system. Atresia or stenosis of the anus and rectum is due to a failure in the normal development of the hindgut. The following are the four major types of rectal abnormalities.

1. This group includes all those conditions in which there is an imperforate anus. The obstruction in these cases is membranous in character.

- 2 In this type of case the anomaly consists of a patent anus but the stenosis of the rectum occurs at a higher level
- 3 In this type of case the anus the sphincter and the lower portion of the rectum are normal but the upper portion ends blindly at a variable distance from the lower pouch
- 4 In this group the anus is imperforate and

the rectal pouch ends blindly some distance above the anus

Diagnosis The diagnosis of obstruction due to rectal or anal malformation is usually quite simple. It is immediately noticed after birth that there is no anal opening or that no stools have been passed since birth. Because these patients present acute obstruction immediately after birth they are gen-

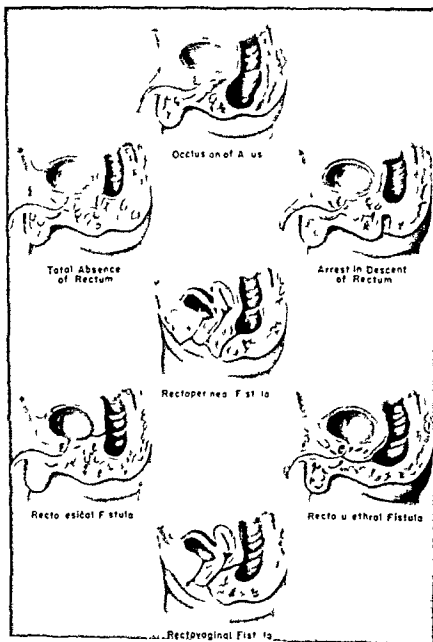


FIG. 221 Congenital malformations of the rectum and anus



FIG. 222 Imperforate anus in a newborn

erally seen by the surgeon in the first few days of life. In an occasional case there will be a passage of meconium through an abnormal exit such as the urethra, the vaginal outlet, or a perineal fistula. In those cases in which there is a normal anus and sphincter with atresia high in the rectum the diagnosis may be overlooked unless an examination is performed radiologically to determine the site of the obstruction. Barium enema is extremely helpful in the diagnosis of such cases. The X-ray study readily discloses the site and degree of the obstruction. In the presence of imperforate anus the X-ray is an important agent in determining the degree of stenosis. By holding the infant upside down the intestinal gas ascends to the nearest point at which the bowel approaches the skin. A film taken with the infant in this position demonstrates the rectal gas bubble and indicates how much of a surgical procedure is required to establish communication between the rectum and skin. However, a wait of 10 to 24 hours for the gas bubble to develop so that it may be used diagnostically not only results in deterioration of the infant but also results in the distended rectum being pulled up further in the pelvis. Accuracy in establishing the level of the rectal pouch is most important since exploration by the perineal route is to be condemned unless one can be sure that the rec-

tal pouch can be reached and a proper repair performed.

Treatment In those cases in which the perineal defect may be an inch or less, perineal continuity of the bowel may be re-established by the perineal approach. In those cases in which the obstruction is merely due to a rectal diaphragm, simple lysis of this rectal diaphragm re-establishes the intestinal continuity.

In those cases in which the rectal pouch ends blindly above the pelvic peritoneum, abdominal exploration is required to close the fistula which may be present between the urethra or bladder and to perform a pull-through procedure to re-establish gastro-intestinal continuity. If the newborn is under 6 pounds or if the general condition of the child will not permit the pull-through procedure, a colostomy with complete diversion of the fecal stream is indicated. By performing a colostomy and leaving the perineum intact, the surgeon later can carry out the pull-through operation and have a normal perineum for establishing a functioning anus rather than one scarred by previous surgery which is both poorly functioning and lacking in control.

Duplications of Bowel as Cause of Obstruction

A segment of reduplicated bowel may cause intestinal obstruction in infancy. A reduplication of the jejunum or ileum may be in the form of a blind pouch with no opening into the normal small bowel or it may be attached at its proximal or distal end to the normal bowel. In an occasional case the reduplicated bowel may be attached at both ends. These reduplications are anomalies which occur on the mesenteric side of the small intestine. In those instances in which duplication occurs as a blind pouch, there may be a marked secretion of intestinal juice into the pouch. This so distends the pouch that encroachment upon the lumen of the overlying normal bowel may occur. This cause obstruction of this overlying normal bowel.

In the management of cases of this type, excision of the reduplicated bowel producing the obstruction may be required. Because of the possibility of injury to the blood supply of the overlying normal bowel, it is necessary to do a primary resection of the overlying normal bowel with the

removal of the reduplicated segment of bowel and its mesentery. Then the continuity of the gastrointestinal tract is re-established by end to end anastomosis. A one or two-loop anastomosis may be used.

Omphalomesenteric Duct

Complete patency of the omphalomesenteric duct has been rarely reported. In a review of 30,000 births at the Chicago Living In Hospital two cases of complete patency of the omphalomesenteric duct were found. Various anomalies which result from faulty or incomplete obliteration of this duct however are relatively common.

This anomaly is said to occur more often in males than in females. In the cases in which the sex has been reported approximately 87 per cent of the infants with persistent omphalomesenteric duct have been males.

The most serious complication as a result of patency of this duct is intussusception or a prolapse of the ileum through the duct onto the abdominal wall. This results in strangulation with gangrene of the involved segment of bowel. Teake described the first case of this type in 1811. The fact that a review of the literature up to 1952 revealed only 35 cases of this type attests to the rarity of this entity. All of the cases reported have been isolated case reports. The age of the infant at the time of prolapse of the small bowel ranged from birth to 7 months with the majority of cases occurring in the first four weeks of life.

An additional type of associated congenital abnormality is a ruptured omphalocele. This is a rather rare lesion and may be associated with other anomalies such as jejunal or ileal atresia. The combination of these two anomalies has been reported with successful treatment by surgical intervention. The association of a ruptured omphalocele and intussusception was noted by the author in a 4 pound premature.

Until recent years the method of treatment was very discouraging. In many of the cases no effort was made to correct the intussusception and the omphalocele. All these early patients died although there was a question of spontaneous reduction and limited survival in two of them.

The operative measures which were instituted

included simple incision of the umbilical ring in an effort to produce a reduction of the intussusception, excision of the omphalomesenteric duct with closure of the ileal defect and occasionally resection of nonviable ileum with the omphalomesenteric duct. The first long term operative survival was reported by Kittle, Jenkins and Dragstedt in 1947. In recent years however there has been a trend toward primary resection and end to end anastomosis as the procedure of choice.

Anomalies of Intestinal Rotation

Anomalies of intestinal rotation may cause intestinal obstruction in infancy and childhood. The most common anomalies of rotation are due to derangements in the second stage of midgut rotation. They rarely occur during the first or third stages. Dott believes that the derangements in intestinal rotation are due to the sudden return through an abnormally large umbilical ring of all that portion of the midgut situated within the cord. As a result there is a failure in the orderly and gradual return of the prearterial loop of midgut to the abdomen. This prevents the proper rotation and arrangement of the intestinal loops within the abdomen. The pathologic arrangements taken by the midgut within the abdomen depend upon which direction the cecum takes after its reduction and return to the peritoneal cavity.

Types of Derangement The following derangements are described.

Nonrotation In this anomaly the cecum passes upward into the left upper quadrant so that the entire colon and cecum are arranged to the left of the midline with the entire small bowel to the right of the midline. The ileum then crosses from right to left to enter the cecum.

Malrotation In malrotation the cecum passes up to the region of the pylorus and becomes attached there or remains in the subhepatic area. As a result elongation and descent are prevented. Because of this there is an elongated narrow mesentery of the fetal type with no points of fixation of the jejunum and ileum except at the narrow duodenocolic angle. Malrotation is occasionally accompanied by the presence of an intra-abdominal or internal hernia. When the cecum, ascending colon and transverse colon are readily seen but the

small bowel appears to be ensheathed by a thin peritoneal membrane in the middle of the abdomen the surgeon should suspect that some internal hernia due to the malrotation is present. In cases of this type, the pre arterial limb of the midgut failed to rotate behind the superior mesenteric artery and as a result was enclosed by its own post arterial mesentery. This type of hernia should be reduced through the hernial aperture and the volvulus untwisted following which the opening of the aperture is tacked closed with two or three interrupted sutures.

Reversed Rotation In reversed rotation loops of small bowel instead of passing from right to left behind the superior mesenteric artery pass from left to right in a clockwise direction. Because of this the cecum comes to lie behind the superior mesenteric artery where it becomes fixed. This anomaly is rather rare.

Premature Fixation Derangements of the third stage of intestinal rotation consist chiefly of the nondescent of the cecum from the subhepatic area to the lower right quadrant. This is believed to be due to too early fixation or in the case of a pelvic cecum it may be due to nonfixation. Under any of these situations volvulus with obstruction may occur.

Types of Obstruction Because of the preceding derangements in rotation or fixation the entire midgut may become suspended from the posterior abdominal wall by a very short mesenteric attachment. The large mass of small bowel and right colon up to the middle of the transverse colon suspended from the posterior abdominal wall by a narrow pedicle may easily undergo torsion. When this occurs the narrow mesentery containing the superior mesenteric artery and vein becomes twisted. This compresses the third portion of the duodenum producing two forms of intestinal obstruction: (1) obstruction of the third portion of the duodenum as a result of compression by the root of the mesentery; and (2) a closed loop obstruction of the midgut caused by volvulus.

Symptoms and Diagnosis The early signs and symptoms of obstruction as a result of derangements in intestinal rotation are those of duodenal obstruction. The infants vomit almost incessantly. Visible peristaltic waves may be noted

Although the vomiting is projectile it does not occur as soon after taking food as in pyloric stenosis and the vomitus contains large quantities of bile. The presence of bile in the vomitus should immediately suggest that the obstruction is not due to pyloric stenosis. Because the obstruction is not complete early in the course of the disease, it may be possible to find some milk curd and bile in the colon washings. When midgut volvulus occurs it usually includes all of the small bowel or all of the small bowel with the cecum and ascending colon up to the middle of the transverse colon. Great emphasis is placed by pediatricians on the emptiness of the right side of the abdomen upon palpation. Strangely enough, in spite of the severity of the twisting of the mesentery of the midgut the blood supply to the small bowel is very often not seriously impaired although there may be venous congestion. Generally the obstruction is not closed at the transverse colon since feces or bloody mucus may be passed per rectum. With the passage of time however circulatory disturbances do occur and strangulation takes place. At this time all the usual physical findings associated with a strangulating obstruction make their appearance.

Treatment It is imperative that this type of obstruction be surgically treated at the earliest possible moment. In such cases (midgut volvulus) while the importance of knowing that the obstruction is due to volvulus must not be underemphasized it is also important to know what the anomalous distribution is. For this reason the abdominal incision should be made in that portion of the abdomen which gives the easiest access to the seat of the pathology. A right paramedian incision of adequate length is usually most desirable. The importance of a generous abdominal incision in the treatment of cases of this type lies in the fact that in no other way can one readily exteriorize the entire small bowel in order to determine the exact pathology present. The mesentery of the small bowel should be carefully inspected to determine the length of its attachment and its position. In addition to recognizing that midgut volvulus has occurred it is essential to know the direction of the twist. In correcting this type of obstruction the mesenteric twist must first be untied until the mesentery can be freed out and the superior me-

enteric artery and its branches made visible. Following this any peritoneal bands producing duodenal compression are carefully cut. This often permits the cecum to fall back into its normal position. In those cases in which nonrotation of the intestine has occurred and the cecum and colon are found in the left side of the abdomen the cecum should be permitted to remain there. Under no circumstances should the cecum be placed in the right lower quadrant and fixed in that position if nonrotation is found to be present. It may be necessary to carefully free the duodenum if it is covered by adhesions of congenital origin. The entire duodenum should be carefully palpated and examined to determine the patency of its lumen because of the occasional association of anomalies in the luminal development of the duodenum with disturbances in intestinal rotation. Obstruction of the lumen of the duodenum should be treated by duodenojejunostomy if possible or gastrojejunostomy. Whenever possible the operation should be of minimal magnitude.

If a mass of small bowel is noted upon opening the abdomen and the cecum is not evident all the bowel must be exteriorized and carefully examined so that the root of the mesentery may be examined and the torsion untwisted. After this has been done cecum and ascending colon should again be carefully examined to determine the presence of congenital bands which should be cut in order to prevent a recurrence of the volvulus. No attempt should be made to anchor the cecum to the right lower quadrant but rather it should be permitted to remain in that portion of the abdomen in which it is found.

After correction of the obvious obstruction a careful search should be made throughout the entire gastro intestinal tract for any other congenital obstructing processes which should be relieved at the time of this surgery.

Volvulus

The presence of volvulus during the first week or subsequent weeks of life has been reported fairly often. At birth however this condition is extremely rare and only isolated case reports have appeared in the literature. Wershub reported a case of volvulus in an infant 20 hours old. In this

case report the obstruction was caused by herniation of several loops of small bowel through a hole in the mesentery permitting a volvulus to develop in the herniated loops of bowel. Other than the hole in the mesentery there were no anomalies in intestinal development found in the gastrointestinal tract. This mesenteric defect was shown to present smooth thick and rounded edges.

A diagnosis of volvulus in the newborn is difficult because the symptoms are very complex and radiologic examination may give data which are difficult to evaluate. In the case reported by Wershub although the infant vomited greenish fluid shortly after birth and continued to vomit being unable to retain fluids it had two normal bowel movements while under observation. Because of this a diagnosis of intestinal obstruction was considered and discarded. It must be remembered that the intestinal tract distal to the point of obstruction will normally empty itself and that one two or even three bowel movements should not obviate a diagnosis of intestinal obstruction. This is particularly the case if other findings of obstruction are present.

Meconium Ileus

Meconium ileus is an obstruction of the small intestine or colon as a result of the lumen being filled with meconium. This should not be confused with meconium peritonitis which is an entirely different clinical entity.

The meconium is a sterile mixture of bile cast off epithelial cells, swallowed amniotic fluid, gastric, pancreatic and intestinal secretions and cholesterol. The obstruction caused by meconium occurs most often in the terminal ileum and has been said to be due to a pancreatic and hepatic insufficiency which may be derived from fibrocystic disease of the pancreas. The obstruction produced by the meconium occurs *in utero*.

Although the cause of the degenerative change which affects the pancreas primarily but may also involve the liver as well as the tracheobronchial glands is unknown when it occurs after birth the familial celiac syndrome develops. Although intestinal obstruction is not a prominent feature of fibrocystic disease of the pancreas when it is found postpartum when this disease affects the infant in

utero the deficient external secretion of the pancreas disturbs the biliary physiology and with the absence of bacterial ferments in the fetal bowel causes improperly digested meconium. Because of this the meconium as it moves down the gastrointestinal tract becomes thick and putty like and in the lower ileum it becomes immovably impacted. When the disease is severe the obstruction of the ileum is as complete as if atresia were present.

Diagnosis When meconium ileus produces complete obstruction of the ileum the signs and symptoms of low intestinal obstruction are found. There is considerable distention. Perforation may occur anywhere along the obstructed gastrointestinal tract. In the colon perforation is uncommon in meconium ileus. When perforation results from the meconium ileus a meconium peritonitis results. This type of peritonitis differs from the acute bacterial type since it is an aseptic chemical peritonitis due to the irritating effect of the meconium in the peritoneal cavity.

Treatment The treatment of meconium ileus is the same as that for intraluminal intestinal obstruction. In former years the outlook was dismal for this type of disease. In recent years recoveries have been reported by surgical intervention in which the bowel is opened and the lumen of the obstructed bowel flushed through with hydrogen peroxide which causes liquefaction of the thick putty like meconium permitting its evacuation. By this means the obstructing meconium may be thoroughly flushed out of the small bowel.

Meconium Peritonitis

Simpson first described meconium peritonitis in 1838 and he reviewed 25 collected cases at that time. However there has been some controversy as to whether all or any of these cases were true meconium peritonitis. A review of the literature regarding the findings in meconium peritonitis produces two large groups:

1. In this group are all of those cases of meconium peritonitis in which there is positive evidence of organic intestinal obstruction. Fifty per cent of the cases reported fall into this group. The lesions producing intestinal obstruction leading to meconium peritonitis vary from congenital intestinal atresia and

hesions, intussusception, volvulus and meconium ileus. The intestinal perforation causing the peritonitis is almost always found proximal to the point of obstruction.

2. In this group of cases reported the meconium peritonitis is also the result of a spontaneous perforation. No point of obstruction anywhere along the gastrointestinal tract is demonstrable, however. Whatever the cause may be the perforation in such cases generally closes before birth and as a result the site of perforation may not be identified even at autopsy.

The treatment of meconium peritonitis consists of the relief of the obstruction and if necessary closure of the perforation. Each case must be treated on an individual basis. Generally no attempt should be made to separate the extensive adhesions found between the loops of bowel. The mortality rate associated with the treatment of meconium peritonitis is high since relatively few cases are successfully treated by surgery.

Fetal Peritonitis

Fetal peritonitis ranks among the unusual varieties of acute intestinal obstruction. It is the least common cause of acute intestinal obstruction in the newborn.

It is believed that in fetal life a rare chronic form of peritonitis may occur which is sometimes ascribed to the absorption of toxins or infections from the mother. Syphilis and tuberculosis have been considered as the most common primary causes. Grille believes that fetal peritonitis is the result rather than the cause of the obstruction and that in practically all instances the cause may be found in some developmental error. Generally, however, in fetal peritonitis there are extensive adhesions between the loops of bowel and as a result of this intestinal obstruction occurs with resulting perforation.

Emsberger reported a case in which it was believed that fetal peritonitis had occurred and was the primary factor in the formation of an intestinal obstruction. He believed that the adhesions formed by this peritonitis prepared the way for the volvulus which obstructed the bowel. He reached this conclusion because at operation the

adhesions were found to be very well formed and were evidently of a chronic or long standing type since they were too dense and fibrous to have formed since birth.

Congenital Bands or Adhesions

Intestinal obstruction due to bands or adhesions in infancy and childhood is rather uncommon. In an occasional case congenital bands are found in the region of the duodenojejunal flexure. In this area incorrectly formed bands may cross the duodenojejunal flexure and produce partial to complete obstruction. In this event the signs of high intestinal obstruction appear early and at the time of operation the releasing of the obstruction involves no more than the cutting of the intestinal bands. In an occasional case conditions are such that a duodenojejunostomy may be indicated in order to bypass the obstructed area.

Malrotation or other rotational anomalies as well as anomalies of fixation during the embryologic development of the intestinal tract may cause congenital adhesions. These may result in either a compression type of obstruction or volvulus.

Obstructing adhesions in the newborn are not always due to rotational anomalies. Rupture of the intestinal tract of the fetus *in utero* or the perforation of the bowel has been reported without congenital obstruction. The escape of meconium produces a chemical peritonitis with resulting adhesions and in some cases encapsulated fluid may occur. The meconium adhesions at times become calcified and may be demonstrated as radiopaque shadows of varying density on a survey film of the abdomen. This occurrence has been found in less than 50 per cent of the cases reported in the literature.

Meckel's Diverticulum

Meckel's diverticulum is a remnant of the vitelline duct which has opened into the ileum. During early embryonic life there is a wide opening between the ileum and the yolk sac. This opening gradually becomes narrowed to form the vitellointestinal duct. If the yolk sac remains patent a Meckel's diverticulum is formed which is sometimes connected to the umbilicus by a cordlike

structure. If the entire duct remains patent an umbilical fecal fistula results.

Meckel's diverticulum is said to occur in approximately 2 per cent of the population and it generally arises from the ileum $1\frac{1}{2}$ to 3 feet from the ileocecal valve and usually opens on the antimesenteric border of the intestine.

Types of Obstruction. Intestinal obstruction is an extremely common complication of a Meckel's diverticulum. The obstruction may occur in the following ways:

- 1 A Meckel's diverticulum may be the starting point for an intussusception which is indistinguishable from intussusception caused by other factors. In 1898 Moron reported a case of polyp and Meckel's diverticulum associated with intussusception. In a series of 160 cases of Meckel's diverticulum associated with intussusception collected by Harkins in 1933 26 per cent of the diverticula contained a tumor. One third of the tumors containing diverticula were found to be pancreatic tissue.
- 2 Meckel's diverticulum may become adherent to the lateral abdominal wall or to an adjacent loop of bowel resulting in a volvulus of the bowel around these fixed points of attachment.
- 3 Meckel's diverticulum may become adherent to the parietal peritoneum in such a fashion that a loop of bowel may encircle it and become strangulated.

Treatment. The treatment in all such cases is early surgical intervention: reduction of the volvulus or reduction of the obstruction and removal of the diverticulum by amputating it at the neck and closing the defect in the bowel in two layers. In some cases primary resection and anastomosis of the loop of bowel to which the diverticulum is attached may be the procedure of choice.

Neurogenic Causes of Obstruction

Neurogenic disturbances in the gastrointestinal tract in infants are capable of producing intestinal obstruction. The lesion diagnosed as Hirschsprung's disease and otherwise known as congenital aganglionic megacolon or ganglionic deficiency

disease ranks as an important cause of obstruction in infancy and childhood

Titel first described changes in the ganglion cells of the myenteric plexus of the colon in a case of congenital megacolon in 1901. He suggested that such changes might interfere with normal peristaltic activity. Following this isolated case reports and studies appeared from 1901 to 1948. These described the absence or degeneration of the ganglia in the constricted portion of the colon as well as the presence of normal ganglia in the dilated segment. Zuelzer and Wilson reported 11 cases of intestinal obstruction in newborn infants due to the congenital absence of nerve ganglia in the colon. The logical surgical treatment for this disease was evolved slowly over a period of 50 years and resulted in the surgical treatment proposed by Swenson and Bill. This treatment was based upon the concept that the pathogenesis of Hirschsprung's disease was due to changes in the myenteric plexus. As a result of this it has been agreed that the proper treatment for this disease consists of a removal of the segment of bowel in which ganglion cells are deficient and the reestablishment of intestinal continuity by the so called pull through operation perfected by Swenson.

Zuelzer and Wilson report a definite familial tendency to Hirschsprung's disease. However other pediatricians have found no familial tendency.

Pathology The essential pathology of Hirschsprung's disease is related to an absence or diminution of the ganglion cells of Auerbach's plexus as well as those of Meissner's plexus. The plexus of Auerbach is a continuous plexus of nerve cells beginning at the junction of the upper and middle thirds of the esophagus and extending to the anus. These nerve cells supply the inner circular and outer longitudinal muscle bundles and lie between these bundles. Meissner's plexus lies within the submucosa. The nerve cells of this plexus are smaller and less frequent than those of Auerbach's plexus. The presence of these aganglionic or ganglion deficient areas in the colon causes an interruption of the normal peristaltic propulsive wave at that point. The failure of these aganglionic areas of colon to contract properly causes functional intestinal obstruction at this

point. As a result of this obstruction there is enormous dilatation and hypertrophy of the colon. Although generally only short segments of rectum and rectosigmoid are affected by such aganglionic areas, in an occasional case the entire large bowel or even a segment of terminal ileum may be involved in the absence of ganglia.

It must not be assumed that ganglion cells are present in the intestine above the point of constriction. It is essential that a microscopic examination of the bowel be made since this is the only certain method for determining the presence or absence of ganglia at any given point. The cessation of ganglia occurs abruptly over a length of 2 to 3 cm. In some cases abnormal appearing ganglia were reported as far as 10 cm. above the area of complete absence of ganglia. Because peristaltic contractions depend upon the nerve plexuses within the wall of the intestine an absence of Auerbach's plexus results in a physiologic intestinal obstruction at that point.

Diagnosis It is often difficult to diagnose these cases because of the great variation in severity manifested by different cases of Hirschsprung's disease. In those instances in which the symptoms are mild there may be no reason to suspect the presence of intestinal obstruction and a diagnosis of mild constipation is usually made. Such infants ultimately develop a megacolon which may be diagnosed correctly as the child grows older. In some cases the symptoms may be of such a degree of acuity that all the characteristic findings of acute small bowel obstruction may appear and it may be difficult to differentiate this disease from atresia of the small bowel.

In a typical case the diagnosis of congenital megacolon (Hirschsprung's disease) may be made very early in life. Signs of low intestinal obstruction become evident rather quickly and if unrelieved the obstruction may become fatal. In such cases vomiting, obstipation and abdominal distention may appear in the first few days of life. Sometimes the infant vomits bile stained material by the second or third day. In some cases obstruction is complete whereas in others the child may be merely constipated. On digital examination in Hirschsprung's disease the rectum is usually found to be empty. In some cases small amounts of

meconium may be passed per rectum, thus meconium may be dry and sticky. Under normal conditions an infant moves its bowels the first day of life and upon digital examination a movement of normal meconium results. This does not occur in the case of a ganglionic deficiency disease. Distention appears fairly early so that intestinal peristaltic waves may be visible.

Radiologic examination can determine the presence of Hirschsprung's disease with a high degree of accuracy. A survey film of the abdomen will generally demonstrate small and large bowel distention with fluid levels. This will help to locate the point of obstruction if the films are made both in the supine and the erect position. Films taken in this fashion may demonstrate gas in the descending colon as well as in the rectosigmoid up to the point of obstruction. By noting the point of transition between normal and dilated bowel a small barium enema may be decisive in arriving at a correct diagnosis. Although this point of transition between normal and dilated bowel may be determined with a high degree of accuracy there may be no correlation between this junction and the presence of ganglia. In some patients a considerable segment of dilated bowel may be found to contain no ganglia. The importance of this point resides in the fact that a resection and anastomosis must be made between two segments of bowel which do contain ganglia.

Treatment. Medical management of Hirschsprung's disease in the newborn is usually unsatisfactory. Accumulated experience has shown that when a diagnosis of ganglionic deficiency disease involving the myenteric plexus has been made it is generally impossible to carry on with conservative medical management in severe cases. Although milder cases can be kept alive with enemas suppositories and drugs no permanent good is accomplished. Resection of the colon with the removal of the portion of the bowel in which the myenteric ganglion cells are absent is the only method of curing such infants.

The preparation of such infants for surgery is important. Prior to operation blood transfusion should be given and the stomach completely emptied by a Levin tube or a long intestinal decompression tube. The latter tube is permitted to pass

on into the ileum to keep the bowel decompressed postoperatively. The abdomen is opened and when a diagnosis of Hirschsprung's disease has been made the condition of the patient determines the procedure to be followed. If the infant is well developed and of average weight with good hydration and electrolyte balance and if there are no other congenital abnormalities resection of the constricted portion of the bowel should be performed. A biopsy study must be made at the time of surgery and all bowel removed up to the point at which normal Auerbach's plexus can be found. After it has been established that the proximal segment contains normal ganglia the anastomosis to the rectum is performed. When the condition of the infant is such that definitive surgery is not warranted or if the colon is acutely obstructed a colostomy is indicated. The Swenson procedure is then best postponed until the infant is one to two years old. The pelvis in the older infants is larger so that exposure is more easily obtained.

Under all circumstances it is imperative that the proximal colon which is brought down to be anastomosed to the rectum have a normal myenteric plexus.

Hernia

Hernia ranks third in importance as a cause of small bowel obstruction in infancy and childhood following hypertrophic pyloric stenosis and intussusception in order of frequency. It has been estimated that approximately 2 per cent of all full term infants may be afflicted with external hernia. This incidence is markedly increased in premature infants so that up to 25 per cent of viable premature infants may present external hernias.

There is considerable confusion in the use of the terms incarceration and strangulation when applied to the problem of hernia particularly in infancy and childhood.

Incarceration. Incarceration includes all cases in which there may be a temporary interference with the blood supply which may result in edema and swelling of the bowel within the hernial sac. Reduction may be obtained by conservative measures in a relatively high percentage of cases and there is no necrosis or gangrenous change in the

bowel wall. It must be emphasized that some of these cases may go on to true strangulation it not reduced. On the other hand the percentage of cases in which strangulation occurs is the result of reduction of an incarcerated hernia is very small.

In most large series of hernial repair performed on infants and children under the age of 12, the greatest incidence of incarceration was found to occur during the first six months of life. After the age of 18 months and up to the age of 6 years there is a steady fall in the incidence of incarceration. After the age of 6 the incidence of incarceration decreases markedly. In a further analysis of the age incidence Thorndike and Ferguson noted that the greatest number of cases occurred during the first three months of life. As would be expected the preponderance of incarcerated hernias was found in males. In addition the right side appeared to be much more frequently involved than the left. Although it may be difficult to prove a possible anatomic reason for this may be the fact that the right testicle generally descends at a somewhat later date than the left the fact that many cases of right sided hernia are associated with an undescended testicle on the same side would seem to bear this out. In such cases a congenital hernia may be caused by the presence of a persistent tunica vaginalis which has not gone through the normal process of obliteration. The preponderance of right sided hernias would then be explained on the basis of an arrest in development during this period of time.

Strangulation The term strangulation is not to be used unless there is sufficient interference with the blood supply of the incarcerated bowel to cause changes in the bowel wall. These hernias are generally not reducible by conservative methods although in an occasional case the hernia may be reduced en masse and strangulation occurs within the abdomen.

Although it is well known that not every case of incarcerated hernia will progress to true strangulation with gangrene it is a recognized fact that it may be impossible in a series of incarcerated hernias to select the one case which will result in strangulation from the ones which will not. During the period of incarceration the bowel may un-

dergo simple edema which may be followed by a marked congestion of its vessels and even an inflammatory reaction in the serosa causing the bowel to become adherent to the hernial sac. It is only when thrombosis of the vessels occurs that resultant tissue necrosis and strangulation of the loop of bowel take place. It must be noted that the circulatory stasis produced by the venous or arterial constriction associated with incarceration proceeds gradually so that unless treatment is instituted in the first few hours of symptoms the process may go on to eventual strangulation.

Treatment The management of this problem in infancy may be classified as either preventive management or therapeutic management.

Preventive Management The preventive management consists of repair of all hernias at the earliest possible moment in order to prevent the possibility of incarceration and strangulation. Only by correcting the hernia before it produces obstruction can one expect to lower the mortality rate to negligible proportions. In such cases the operation is a simple one the risk is minimal and the morbidity is small. The results are generally gratifying.

Most physicians are very reluctant to subject the newborn to repair in the erroneous belief that intestinal obstruction and strangulation rarely occur with such hernias. In recent years many articles have appeared describing strangulated hernia in 1 month old infants necessitating resection and end to end anastomosis. From this it would seem that a more aggressive attitude would be desirable and that all such hernias should be repaired as early as possible to avoid the serious consequences of intestinal obstruction and the possible hazards of strangulation requiring bowel resection with its attendant dangers.

Therapeutic Management The therapeutic approach to the problem of those infants admitted to the hospital with incarceration of less than four hours duration consists of an attempt by means of sedation to relieve spontaneous reduction of the hernia. Trendelenburg's position and gentle taxis may be employed. It should be emphasized that it is possible to reduce en masse a strangulated loop of bowel which goes on to perforation within the peritoneal cavity. These cases however, which

are incarcerated for four hours or less may be spontaneously reduced by such simple maneuvers and rarely cause difficulty. This is especially true if the hernia is spontaneously reduced by the use of a sedative and the Trendelenburg position but without the use of taxis. Following this an elective hernial repair may be performed several days later when the patient is better prepared for surgery.

If the hernia is not reducible within one hour following these conservative maneuvers or if the surgeon believes that nonoperative reduction is contraindicated, the patient should be prepared for surgery. In such cases it is desirable to pass a Levin tube and suction out the gastric contents. The suction should be continued throughout the operation and for the first few postoperative days in order to prevent the intestinal distention associated with air swallowing. This will prevent regurgitation of the gastric contents during and after surgery which may result in aspiration pneumonia or atelectasis. Intravenous fluids should be started immediately in order to keep the electrolyte and water balance at normal levels. In any case at operation it is of the greatest importance that the contents of the hernial sac be inspected for viability before being returned to the peritoneal cavity. In no case should the contents of the hernial sac be permitted to fall back in the peritoneal cavity until carefully inspected for signs of strangulation. The presence of a bloody fluid or blackish fluid within the hernial sac is highly suggestive of compromise of the bowel circulation which has been incarcerated.

In the corrective treatment of inguinal hernias in infancy and childhood the sac should be dissected free, opened, the contents returned to the peritoneal cavity if not strangulated and the neck of the sac ligated. This is adequate treatment for such cases. There are many, however, who still prefer the Ferguson procedure as a reparative measure.

Types of Hernia That May Cause Obstruction All types of hernia are capable of causing intestinal obstruction. Some types however are more prone to do this than others. The following are the most commonly encountered types of her-

nias associated with intestinal obstruction in infancy and childhood.

Omphalocele Omphalocele or umbilical hernia is relatively uncommon. The umbilical ring shows progressive contraction up to 18 months. For this reason a conservative type of treatment is indicated in the newborn. Adhesive strapping is of little or no value and only succeeds in irritating the skin. Since most umbilical hernias generally disappear spontaneously within the first year of life elective hernial repair should be delayed until the child is over a year old. If an operation must be performed in such cases a curved sub-umbilical incision is used in order to preserve the cutaneous umbilicus.

Congenital Internal Hernias Congenital internal hernias such as paraduodenal hernias are manifestations of malrotation. In such cases the hernia should be reduced into the peritoneal cavity and the opening into the paraduodenal fossa closed with interrupted sutures of nonabsorbable suture material.

Congenital Diaphragmatic Hernias Congenital diaphragmatic hernias may occur as a result of a defect in the diaphragm with extension of the splenic flexure of the colon into the left chest. At times the small bowel and stomach may also be found in the chest. Diaphragmatic hernia in the newborn infant was a much neglected anomaly until recent years. It is not uncommon however. Without treatment the mortality rate approaches 90 per cent. Reviewers on this subject report that 75 per cent of the patients with congenital diaphragmatic hernia died before the end of the first 48 hours of life. A correct diagnosis is not difficult in cases of this type. These infants have attacks of cyanosis especially during or shortly after feeding. There is a tendency to vomit. As a result of the encroachment on the chest cavity by the abdominal organs the respiratory rate may be accelerated. On examination the heart may be found to be pushed to the side opposite the hernia with tympany to percussion in the region of the hernia. Auscultation over the thorax may reveal a clear sound. An X-ray of the chest without the use of contrast media usually makes the diagnosis obvious.

Surgical treatment is an emergency procedure in

the newborn with this type of hernia. Never transfuse these infants before operation as they already have a high hemoglobin and may develop right heart embarrassment as a result of the unnecessary transfusion. Surgical treatment is essential but should be delayed until fluid and electrolyte balance has been reached. A transthoracic or abdominal approach may be utilized in the repair of these lesions. Harrington reported two cases in which a transthoracic approach was used but in which because of intra-abdominal adhesions it was necessary also to open the abdomen. Both Ladd and Gross advocate the abdominal approach.

Inguinal Hernia Inguinal hernia, the most common of all types of hernia in infancy and childhood, may be readily overlooked. Any examination of a child with intestinal obstruction should include a careful search for incarcerated inguinal hernia. It is only when the hernia is obviously incarcerated that one can be reasonably certain that some coexisting intra-abdominal anomaly is not present producing the obstruction.

The one single symptom common to every type of obstruction in infancy and childhood is vomiting. Since a history of colicky pain in the abdomen is not obtainable, the vomiting is of great importance in suggesting obstruction. The character of peristaltic sounds on auscultation of the abdomen in an infant is relatively unimportant when compared with the extreme importance of this diagnostic sign in adults. This is due to the fact that an orderly and regular pattern of peristaltic activity may not be established for several weeks or months in an infant. Because of this, obstructions of the newborn result in intestinal distention relatively early and the stomach is notably capable of remarkable distention due to swallowed air and ingested fluid. In the management of all inguinal hernias, the sac should be opened, the hernial contents examined for circulatory impairment and if none is present, the contents reduced into the peritoneal cavity. The hernia may or may not be repaired at that time depending upon the condition of the patient. In the case of inguinal hernia, a Ferguson type of hernial repair is the procedure of choice. Recent writers on the subject, however, are unanimously agreed that simply ligating the hernial sac at its neck and removing the redundant

sac is all that is required since the lesion is due to the congenital persistence of the tunica vaginalis testis. No further repair measures are required in such cases.

Postnatal Causes of Obstruction

The postnatal causes of intestinal obstruction in infancy and childhood are relatively few in comparison with those of congenital origin. The following types are entirely postnatal with no congenital factor being present.

Adhesions

Adhesions as a cause of intestinal obstruction may be the result of surgery upon the appendix or as a result of appendicular disease, either appendicitis or local peritonitis. In a review of intestinal obstruction in children, Penberthy, Noer and Benson found that in 85 per cent of the cases the ileus was the result of appendicular disease in one form or another. In such cases it is inevitable that



FIG. 223. Acute intestinal obstruction produced by adhesions following appendectomy. Note the fluid levels and the marked degree of small bowel obstruction.

intestinal obstruction may occur as an early or late complication of the primary disease process.

The management of adhesions in the very young differs little from that employed in adults.

Intussusception

Intussusception was recognized as a pathologic entity early in the 17th century. Nyborg in his paper on intussusception in infants reported that the French surgeon Francois Kochan in 1627 was the first to suggest that intussusception be excluded from the collective term *ileus* which was used for all forms of obstruction. In addition Nyborg reported that the first specific clinical observations on this disease were made by Kuhn in 1702. John Hunter in 1789 was one of the earliest to describe the clinical characteristics of this condition. The term intussusception was introduced by Rokitan sky in 1837. He also introduced the terms *intussusceptum* and *intussusciens*. The first thorough analysis of the literature on the subject was made by Feichtenstern in 1876. He described the various types of intussusception as enteric, ileocecal, ileocolic, and colic. The first successful operative reduction of intussusception in a child was reported by Jonathan Hutchinson in 1874. From that time onward improvements in the surgical technique, the introduction of antibiotics, and earlier recognition and treatment have resulted in a reduction of the operative mortality rate from 59 per cent to the 27 per cent reported in 1948.

Incidence. The incidence of intussusception has been variously reported. McLaughlin noted that 0.16 per cent of all admissions of infants and children was due to intussusception. Ladd and Gross reported an incidence of 0.3 per cent.

Pathologic Anatomy. Acute intussusception is primarily a disease of infancy and early childhood. It occurs most commonly in infant under the age of two and is the most common cause of acute intestinal obstruction for this age group. Seventy-five per cent of all cases reported appeared to occur during the first two years of life, with half of these occurring during the first nine months of life. The type of feeding, whether breast or bottle, appears to have little influence.

An intussusception consists of an intussusciens or outer encircling or enveloping layer and

an intussusceptum which is that portion of the bowel within the ensheathing layers. There are many varieties of intussusception. The varieties occurring within the small bowel are called enteric. Those which occur within the colon are called colic. According to the anatomic involvement there may be ileocecal, ileocolic, or cecorectal varieties. In addition there may be the rare retrograde involvement, either ceco-ileal or colico-ileal.

Etiology. Intussusception may be divided into two large groups. In the first group are all those cases in which the predisposing factor is mechanical. The second group consists of all those cases in which no organic cause for the intussusception can be found.

Mechanical Causes. In this group one finds such lesions as polypoid growths on the mucosa, lipomas, Meckel's diverticulum, abnormality of the mesentery, disproportion in size between the small intestine and the large intestine, hypertrophied Peyer's patches, and appendical disease.

It is not unusual to find aberrant pancreatic tissue within a Meckel's diverticulum which furnished the nidus for intussusception. This aberrant pancreatic tissue may also be found as whitish, opaque, lobulated tumor masses anywhere along the gastro-intestinal tract, being most commonly found in the submucosa. The next most common point of location is intramucosal (within the muscle layers) and the least common is subserosal. There have been many cases reported of such aberrant pancreatic tissue furnishing the source for intussusception unassociated with a diverticulum. In such cases the pancreatic tissue furnishes a space-occupying lesion within the bowel wall which results in a nidus for the intussusception.

Familial polyposis is a rather rare cause of intussusception in infancy and childhood. A review of the literature and a report of cases by Williams and Williams in 1949 revealed only four other recorded cases of intussusception due to the familial occurrence of such tumors in the small intestine. In a remarkable family reported by Sharber, Roan, Haggard, and Floyd, three members underwent multiple operations for intussusception due to adenomas of the small intestine. Nine resections were performed on three members of the family.

GASTRO-INTESTINAL OBSTRUCTION AND PREGNANCY

Intestinal obstruction as a complication of pregnancy is relatively uncommon. It may occur with any of the three phases of pregnancy—before labor, during labor, or either early or late during postpartum. Estimates of the occurrence of intestinal obstruction during pregnancy are extremely variable. Incidences as low as 1 case in 66 000 pregnancies and as high as 1 in 7000 have been reported. Eleven cases of intestinal obstruction associated with pregnancy were found in 13 442 obstetric patients admitted to Grace Hospital during the 5 year period from 1946 through 1950. This is an incidence of 0.08 per cent. Of this small group of 11 patients, three cases (27 per cent) occurred during the puerperium.

DIAGNOSIS

Intestinal obstruction is often difficult to diagnose in the pregnant woman because many of the associated symptoms, such as vomiting, pain, obstipation, and distention, are also found in the course of a normal pregnancy. The difficulty in diagnosing intestinal obstruction is further complicated during the latter months of pregnancy by the fact that many women complain of pain in the abdomen during the last trimester of pregnancy. Furthermore, the pains associated with labor are difficult to distinguish from those produced by intestinal obstruction. However, in such cases, auscultation of the abdomen will lead one to a correct diagnosis by revealing the correlation of borborygmus with the cramping pain. Borborygmus does not occur with labor pains and is there-

fore an extremely important diagnostic point in such cases.

Further differentiation between obstruction and pregnancy is possible. For example, although vomiting is a very common finding in the early months of pregnancy, it is usually not expected after the fifth month. Consequently, vomiting after the fifth month should lead one to suspect the presence of an obstructive process. Similarly, although a colicky abdominal pain is generally common to both pregnancy and intestinal obstruction, the association of borborygmus or sound with this pain is a good indication of obstruction. An additional clue is the presence of a bloody vaginal discharge in those cases in which the colicky pain is due to the pregnancy. This should always be looked for in the pregnant woman. Unfortunately, abdominal distention and constipation are so common during pregnancy that they are of very little value in the differential diagnosis.

The most significant sign of intestinal obstruction in pregnancy is the presence of hyperperistalsis, demonstrated by high pitched intestinal sounds associated with colic. This is almost pathognomonic of small bowel obstruction. A survey film of the abdomen may be of great value by demonstrating loops of small bowel filled with gas and fluid, which are typical of small bowel obstruction. In any event, when faced with complaints of obstipation, pain, distention, and vomiting, the obstetrician should look for evidence of bowel obstruction before attributing the symptoms to the pregnant state itself.

CAUSES OF OBSTRUCTION

Intestinal obstruction associated with pregnancy may be due to the same etiologic factors which cause obstruction in the nonpregnant individual. However, the presence of the enlarging uterus alters the relative importance of the usual factors. The common causes of intestinal obstruction with pregnancy follow.

Adhesions

Adhesions constitute the most common cause of intestinal obstruction in pregnancy. In late pregnancy, the adhesive bands which result from

previous surgery on pelvic organs may become so stretched that they sharply angulate or kink the bowel thus obstructing it. In addition to the mechanical obstruction produced by adhesions, volvulus may occur occasionally when a loop of bowel which is adherent to a pelvic organ or to the abdominal wall becomes twisted on its axis as a result of pressure from the enlarging uterus. The enlarging uterus pushes the bowel into the upper abdomen. In many instances the enlarging uterus so kinks and distorts the bowel that it converts a partial obstruction into a complete one. This is particularly apt to occur in those cases in which a loop of small intestine has become adherent to the round ligaments or is attached to the uterus posteriorly subsequent to a Baldy Webster type of suspension. Although with a nonpregnant uterus this may not interfere with the function of the small bowel during pregnancy, the enlarging uterus distorts and angulates the adherent loop of bowel thus obstructing it. In the management of intestinal obstruction from this cause surgical



FIG. 224 Small bowel obstruction as a result of adhesions in a fourth-month pregnant woman. Note the small bowel distention. Note also the fetus in the pelvis. In this case the radiologic findings were those of ileus whereas the clinical findings were those of mechanical intestinal obstruction. Following the clinical indications and performing the proper surgery the patient was cured. This is an example of the fact that intestinal gas patterns may be fallacious in the diagnosis of mechanical intestinal obstruction.



FIG. 225 Four month pregnancy with small bowel volvulus completely obstructing the gastroduodenal tract. Note enlarged and bony parts of early fetus. Small bowel is distended and fluid levels are obvious.

intervention is indicated at the earliest possible moment. Such intervention may consist simply of releasing the adhesive bands and separating the intestinal coils from the area to which they have become attached or it may involve a resection of strangulated bowel and end to end anastomosis.

Intussusception

Intussusception associated with pregnancy is rather uncommon. In a review of the literature on intussusception during pregnancy covering the period from 1870 to 1937 Chaffin Mason and Slemmons found 19 patients reported to which they added a case of their own bringing the total to 20 cases. In these cases of intussusception associated with pregnancy high intestinal obstruction was found to be far more dangerous than low obstruction because of the speed with which electrolyte imbalance occurred. Of the 20 cases reported up to 1937 15 patients died a mortality rate of 75 per cent. The fetal mortality rate in this group of 15 patients was 75 per cent live babies being born to a quarter of the moribund patients. In two patients intestinal obstruction developed during labor. In this small group both mothers died (100 per cent) while the fetal mortality rate was 50 per cent. From these early statistics it should be apparent that intussusception associated with pregnancy is an extremely serious problem for both the mother and the unborn child. *Auto amputation* in which a necrotic segment of bowel was passed per rectum occurred in 6 of the 20 cases reported. In four of these six cases the auto amputation occurred during or prior to the sixth month of pregnancy and of this number three were delivered of viable infants at term.

In recent years there has been a considerable improvement in the maternal and fetal mortality statistics because of our tremendously improved methods of handling this disorder. Consequently many of the older statistics while of interest must be discarded in the light of newer knowledge although the fundamental principles of management remain essentially the same.

Pyde described an unusual case of intussusception in a postpartum patient. This woman had passed 6 inches of intussuscepted bowel and was discharged from the hospital two weeks later

apparently well. She had excreted the intussusception and had successfully undergone auto amputation. Two weeks after her discharge from the hospital however she was readmitted with obvious acute intestinal obstruction requiring emergency surgery. At operation the obstruction was found to be due to an inflammatory reaction and obstruction at the site of the auto amputation. The obstructed bowel was resected by the surgeon and the patient recovered.

Hernia

It has been found that most hernias are made larger by the relaxation typical of the pregnant state. The enlarging uterus generally pushes the abdominal contents upwards as it expands and as a result the danger of obstruction through a femoral or inguinal hernia is greatly reduced. When there are adhesions between the bowel and the hernial sac however intestinal obstruction may be caused by the traction exerted. In those cases in which incarceration occurs the procedure should be the same as it would be for the nonpregnant individual. Surgical treatment should be instituted at the earliest possible time. Strangulation of a loop of bowel in a hernia is relatively uncommon during pregnancy because such loops of bowel are pushed away from the inguinal and femoral regions as well as from incisional defects by the enlarging uterus.

Volvulus

Volvulus of the cecum although rather uncommon in association with pregnancy is by no means rare. Small bowel volvulus on the other hand is very rare with only an occasional case reported in the literature. Kerr and Kirkaldy Willis report the occurrence of small bowel volvulus in an 8 months pregnant woman who delivered a normal child. Volvulus like intussusception is extremely difficult to diagnose in the advanced stages of pregnancy because the enlarged uterus prevents satisfactory abdominal palpation. However the presence of this enlarged uterus does not interfere with the radiographic study which will facilitate accurate diagnosis. The management of this type of obstruction is purely surgical and must be initiated as soon as the correct diagnosis is made.



FIG. 226 Volvulus of the cecum in the early postpartum period. This patient was operated upon and cecostomy as a fixation was performed.



FIG. 227 The same patient as in Figure 226. Barium enema demonstrated a volvulus of the cecum on its transverse axis.

Small Bowel Tumors

Tumors of the small bowel may develop prior to or during the pregnant state. The slowly growing tumors may be responsible for intestinal distention and may cause signs of a partial small bowel obstruction. Rapidly growing tumors may cause complete obstruction with the signs and symptoms of acute small bowel obstruction. The accepted method of bowel resection and end to end anastomosis should be followed in all cases of this type. The basic considerations with reference to the viability of the child must be considered in the management of such cases.

Infections

Various infectious processes may produce paralytic ileus as well as mechanical obstruction in the pregnant state. Among these the most common is acute appendicitis. The types of obstruction resulting from acute appendicitis in the pregnant state differ little from those produced in the non-

pregnant patient. Paralytic ileus may result from a localized or generalized peritonitis and at times mechanical intestinal obstruction is produced by an appendical abscess. The tendency toward miscarriage or abortion which often occurs in the presence of peritonitis should be watched for. Conservatism is the general rule in the management of intestinal obstruction or paralytic ileus associated with appendical inflammatory processes in the pregnant as well as the nonpregnant patient. In those patients however in whom an appendical abscess developed at or near term delivery of a live baby from below should be considered.

The pregnant woman is more susceptible to poliomyelitis than is the nonpregnant woman. In this event intestinal intubation frequently will carry the patient through the period of intestinal distention resulting from the ileus permitting the delivery of a normal viable child at term.

Renal disorders are particularly common in the last trimester of pregnancy. These may cause para-



FIG 228 Note the tremendous intestinal distention particularly in the region of the stomach. Note also the presence of an eighth month fetus. This patient M D age 27 white female was admitted to the hospital with a diagnosis of intestinal obstruction. On the survey film the radiologist made a diagnosis of volvulus of the cecum.



FIG 229 The same patient as in Figure 228. A Levin tube was passed into the stomach but suction was not applied. As a result notice the tremendous distention of the stomach with a fluid level. Peculiar suspicion was not applied the erroneous suspicion that volvulus of the cecum was present was still entertained.

lytic ileus late in pregnancy. Here again the management is conservative and the treatment should be directed to the relief of intestinal distention as well as to the management of the infectious process in the kidney tract.

Obstructions of the Colon

Obstruction of the colon may be produced by the pregnancy itself. In such cases the obstruction is low involving the sigmoid colon and distention dominates the picture. The symptoms differ considerably from those found with volvulus where the pain is severe. When the obstruction is directly due to the pregnancy, barium enema shows the site of the obstruction to be at the rectosigmoid. Why pressure from the pregnant uterus should cause obstruction in a small percentage of cases and not in others where the same factors appear

to be present has not been determined. It is suspected that the pressure of a pregnant uterus itself would not be sufficient to produce obstruction in a normal individual but rather that other factors are required—an atonic colon due to chronic constipation or a megacolon. Additional factors may be retroflexion of the uterus, hydrocephalus of the fetus, hydramnios or adhesions about the sigmoid colon which bind it down so that it can not be normally displaced by the enlarging uterus and as a result is compressed against the lower pelvic brim.

When pregnancy is the cause of the obstruction it has been suggested that the patient be placed in the knee elbow position as a means of relieving the pressure of the pregnant uterus against the atonic distended colon which is responsible for the obstruction. Several cases of this type have



FIG. 230 The same patient as in Figures 228 and 229. The application of suction to the Cantor tube which had been passed into the stomach brought complete decompression. It was thus obvious that the patient was suffering from tremendous gastric dilatation and paralytic ileus as a result of poliomyelitis and that volvulus of the cecum was not present.

been reported in which this method of positioning was used successfully to relieve the obstruction.

In 1944 Kohn, Briele, and Douglass reviewed the literature and reported 79 cases of volvulus of the cecum in pregnancy. Nineteen of these were found to involve the right colon. Feuth and Obladen illustrated radiographically the manner in which the enlarging uterus so displaces the mobile cecum as to cause volvulus. However, Pratt and Fallis believe that volvulus is caused during pregnancy not so much by mechanical means as by the abnormal peristaltic activity accompanying the vomiting of pregnancy.

Obstruction of the cecum may sometimes occur as a result of its incarceration in a sliding hernia on the right side. This is rather rare, however.

Carcinoma of the colon as a cause of obstruction in women during their childbearing period is un-

common. In a review of the literature in 1945 Linner, Hunt, and Dixon reported 62 cases of carcinoma of the colon with pregnancy. Of these the percentage in which acute intestinal obstruction occurred was small.

Abdominal Pregnancy Causing Obstruction

Abdominal pregnancy is an extremely uncommon complication of pregnancy associated with intestinal obstruction. The rarity of this condition is shown by the fact that only three such cases were recorded over a 10 year period in a large metropolitan hospital in Detroit. Some degree of bowel obstruction is invariably associated with abdominal pregnancy. In an occasional case the obstruction may be complete. Most abdominal pregnancies are operated upon relatively early in the course of gestation. In an occasional instance the diagnosis is not made until after the viability of the fetus has been established. Unfortunately it is seldom possible to obtain a live baby. Two factors appear as the cause of intestinal obstruction in abdominal pregnancy:

1. The matting of loops of small bowel by the attached placenta.
2. The fetus itself, which constitutes an abdominal tumor and may produce obstruction by compression.

Lithopedion is a late sequel of abdominal pregnancy. A case of this type was reported by Glass and Abramson in which volvulus of the cecum occurred. These authors reported intestinal obstruction due to volvulus and perforation of the cecum as a result of the adherence of a mobile ascending colon to a retrocolic lithopedion. The point of twisting of the volvulus arose at the site of adhesion of the ascending colon to the retrocolic lithopedion. This type of obstruction is extremely rare.

MANAGEMENT OF OBSTRUCTION IN PREGNANCY

The management of intestinal obstruction associated with pregnancy presents a very difficult problem. The surgeon is confronted with the problem of both the patient and the growing fetus. From the obstetric point of view, there are definite rules for managing the pregnancy in the



FIG 231 A survey film of the abdomen showing a lithopedion lying in the right lower quadrant of the abdomen with multiple dilated loops of small and large intestine visible. Cecal volvulus was the result of an adhesive band from the lithopedion becoming adherent to a loop of small bowel and a resultant twisting of this loop effectively obstructing it.

presence of an obstruction of the bowel. While each case must be handled individually, such factors as the length of gestation, the nature of the obstruction, the general condition of the patient, the condition of the fetus, and the possibility that delivery from below or cesarean would deliver a live child should always be considered.

Early in the course of pregnancy, the obstructive process may be corrected and the pregnancy permitted to continue undisturbed. After the period of viability has been reached, however, the procedure employed depends to a great extent upon two prime considerations—obtaining a viable child and the danger of the obstruction to the

pregnant woman. If it seems fairly certain that a viable child can be obtained by emptying the uterus from below, this should be done before instituting treatment for the obstruction. In some cases, when a mechanical obstruction has been caused by the enlarging uterus which has produced stretching of adhesive bands to the abdominal wall, delivery of the pregnant uterus alone may be sufficient to relieve the obstruction. In those cases, however, between the fourth and sixth month of gestation when the fetus is approaching the viable stage, a high degree of obstetric as well as surgical judgment is required for a successful outcome. In such cases, if one feels reasonably certain that a strangulating obstruction is not present, which as we have pointed out before is no mean feat, an attempt should be made to treat the obstructing mechanism by intestinal intubation. If the intestinal decompression tube progresses satisfactorily and the bowel becomes decompressed, a reasonable attempt should be made to continue the pregnancy in order to obtain a viable child. This course of action should be interrupted at any point at which the presence of strangulation of the bowel is even remotely suspected, and immediate surgery should then be instituted. Furthermore, if the progress of the case is not considered satisfactory by the attending surgeon, this period of conservative management may be interrupted at any time to correct the obstructive process surgically. In many cases, however, the conservative approach using the long intestinal decompression tube makes it possible to carry the patient well into the period of viability. However, the surgeon who institutes this form of management assumes a far greater responsibility than the one who elects early surgical intervention as a routine measure in all cases.

In those patients in whom pregnancy is far advanced, the technical difficulties associated with abdominal operative procedures may be greatly reduced if the pregnant uterus is emptied prior to surgical intervention. This should be done from below, if possible. In any case in which an abdominal hysterotomy is performed to remove a viable fetus, this procedure should precede a bowel resection or any other bowel surgery in order to avoid contraction of the uterus. No cut and

dried rules of procedure can be instituted in the management of such cases. At times it may be possible to do a resection of the colon and end to end anastomosis without disturbing the pregnant uterus particularly when the pregnancy is in the early months. Every effort should be made to preserve the viable fetus if this can be done without endangering the life of the mother.

At operation in intestinal obstruction complicating pregnancy the surgeon is confronted with a twofold problem—relief of the intestinal obstruction and what should be done about the pregnancy. In all cases the surgeon must direct the surgical procedure in order to give the mother the best possible chance of survival.

The treatment of intestinal obstruction associated with pregnancy must be more vigorous and active than would be the case under normal conditions. Since generally nothing is gained by conservative management once a diagnosis of mechanical obstruction has been made surgical intervention as soon as possible after correction of electrolyte imbalance and dehydration is the procedure of choice. As a result of instituting this management policy at Grace Hospital the mortality rate associated with intestinal obstruction in pregnancy in the small group of 11 cases was 9 per cent (one death). This mortality rate is appreciably below the 14 per cent average mortality rate for the management of all types of intestinal obstruction during the same period of time.

The risk to the fetus is great in any case of obstruction associated with pregnancy. In a review of 300 pregnancies complicated by ileus of various kinds, Essen Moeller reported that the fetus died in from 66 to 75 per cent of the cases. In his statistics he included still births, infant deaths after spontaneous delivery and deaths due to premature termination of pregnancy by artificial means. In many series of cases reported the

fetal mortality rate ranged from 50 to 75 per cent. These statistics attest to the seriousness of obstruction insofar as the fetus is concerned.

Although the risk to the fetus is considerable the risk to the mother may be even greater if the obstructive mechanism is not properly treated. The chances of postoperative miscarriage or some other fetal complication can never be accurately prognosticated at the time of surgery. For this reason the duration of the obstruction, the condition of the patient and the character and the magnitude of the surgical procedure required to correct the obstructing process must be considered in deciding what must be done about the fetus.

It is generally agreed that after any major surgical procedure upon a pregnant woman moderate sedation should be maintained for several days. There are many gynecologists who suggest the daily use of 10 mg. of progesterone given intramuscularly. In those cases in which surgery is performed during the first trimester of pregnancy the dose of progesterone should be doubled. There are many obstetricians who advocate the daily use of 10,000 to 25,000 units of estradiol along with the progesterone in any pregnant patient subjected to surgery. Other obstetricians advocate the use of large doses of stilbesterol alone. There is general agreement that early ambulation is contra-indicated in this group of patients unless for some constitutional reason.

In an occasional case of acute intestinal obstruction particularly when there is marked intestinal distention and the point of obstruction is found in the pelvis, cesarean hysterectomy may be required before the obstructive process can be adequately dealt with. Although the need for this surgical procedure seldom arises it should be considered in any case in which adequate exposure cannot be obtained even after the delivery of a live baby by cesarean section.

OBSTRUCTION IN THE AGED

In our classification of the aged we include all those individuals ranging from 60 years of age up with an average age of 72 years. The condition found in the aged has been defined as a physiologic state of maturity when as is often true pathologic changes have complicated the senile state. Under this definition we should not attempt to restore the patient to his pre senile state of normalcy but should simply endeavor to bring him to his normal state of maturity. It is generally agreed that the actual age of the individual is of little importance. The physiologic and pathologic status of his vital organs must be our index in determining his ability to withstand treatment. A dissipated individual of 40 could well be a more serious risk than a well preserved man of 70 years.

It is very difficult to set a line of demarcation between middle age and old age. Most people believe that a man is as old as he feels. Common usage, however, has placed those individuals beyond the age of 60 in the old age group, taking into account the fact that it is the physiologic rather than the chronologic age of the individual that really counts. With the boost in the average span of life from 49 years in the 18th century to the present life expectancy of 68 years, the number of elderly patients with intestinal obstruction has increased and may be expected to continue to increase.

Our former concept that a patient is too old for surgery is no longer tenable. Believing that reckoning age on the basis of years alone was a very fallacious method, Sir James Paget described two types of old people. He noted that those old people who are thin and tough and physically ac-

tive with clear voices, bright eyes, good stomachs and strong wills are good surgical risks, being able to bear almost any operation except those of the greatest magnitude. However, those aged patients who are weak, soft skinned, with poor pulses, bad appetites, poor muscular development and such poor digestive powers that in an emergency they cannot be well nourished must be considered poor surgical risks. In addition, Paget noted that a heart or excretory system that has carried the individual successfully through 70 years of active duty can generally be expected to come through the major surgical procedure without trouble. Recent statistical reports demonstrate that in spite of the increased risk of surgery in the aged, well planned surgical procedures will invariably carry the aged patient successfully through major surgery. This concept is in direct contrast to that of former years when surgery was performed in the aged only in an extreme emergency and elective procedure were avoided.

ASSOCIATED PROBLEMS IN THE MANAGEMENT OF THE ELDERLY OBSTRUCTED PATIENT

There are certain physiologic as well as pathologic risks associated with aging, so that safe surgical intervention in the aged patient requires more detailed attention and additional care. Specific diseases of the circulatory system such as the myocardium and disorders of the kidney and liver offer unwelcome risks. In the aged there may be varying degrees of coronary sclerosis with myocardial fibrosis and very often myocardial infarct in various stages of healing may be present. Because of this it is not unusual to find that the

myocardium in the aged may be weakened and its reserve impaired. It is therefore not unusual for the mechanisms of cardiac death to be due to congestive heart failure as a result of exhaustion of the cardiac reserve, sudden coronary occlusion or ventricular dysfunction as a result of interference with the conduction system. Peripheral circulatory collapse or massive pulmonary embolus may occur. Because of this, particular attention must be given to the cardiac status of all elderly patients subjected to surgery. The liberal use of digitalis under the supervision of an internist will do much to prepare these elderly patients for surgery. In increasing the myocardial reserve, the dangers of pulmonary congestion as ocurred with a weakened myocardium are largely avoided and as a result the chance of terminal pneumonia is greatly decreased. In addition, great care should be taken in giving intravenous fluids to the aged because of the danger of overloading the circulation with fluid.

These patients do present many of the diseases of senility in a chronic or an advanced form along with the normal deterioration of age. The factor of deterioration which is a part of the aging process causes the problem of urgent surgery in the older age group to assume its special character. The additional diseases present in the aged in a chronic or in an advanced form provide an additional hazard in the management of acute obstruction. When a surgical emergency such as acute intestinal obstruction arises in this age group there is often little opportunity to improve the patient's physical status or to correct fundamental organic defects. Obesity, malnutrition, cerebrovascular accidents, diabetes, vitamin deficiency, states of hypoproteinemia, arterio-sclerotic diseases, renal disease and many other disease processes may be encountered. Because these disease processes may have been present for a long time, it may be very difficult to make an early diagnosis of intestinal obstruction. Many of the primary chronic diseases inherent in the aged may mask or mimic the early signs and symptoms of acute intestinal obstruction. In addition, the pre-existing disease process and the deteriorated condition of the patient may also result in postoperative complications which probably would not otherwise occur. Many of

these older patients are particularly prone to pneumonia, wound disruptions as a result of malnutrition and infection. After successful surgery, some of the aged patients succumb to cerebrovascular accidents, coronary occlusion, uremia or cardiovascular renal disease.

EXAMINATION AND DIAGNOSIS

The examination of any patient in this age group must begin with a digital rectal examination as well as a sigmoidoscopic examination. Following this, a survey film of the abdomen should be taken. This is then followed by a barium enema X-ray after the survey film has been examined to determine the presence or absence of gas-filled bowel loops and their locations as well as to decide whether the small or large bowel is distended with gas. A barium enema is of great value in determining the type of obstruction and its location so that a direct approach to the obstructive process can be made by the surgeon with a minimum of abdominal exploration. The radiologic diagnosis may be difficult because when the colon is filled with fecal material there is no gas-distended large bowel. As a result, a diagnosis of obstruction on the basis of the survey film alone can not be made. The presence of a fecal impaction is apt to lull the unwary into assuming that this is the sole obstructing mechanism present so that a further search for more proximally situated obstructions may not be carried out.

A diagnosis of intestinal obstruction in old people as well as the determination of its etiology may present great difficulties. Since many of these patients are chronically constipated, the absence of bowel movements for several days can easily pass unnoticed. Similarly, a complaint of some abdominal discomfort may be overlooked and the combination of abdominal pain with constipation is quite likely to result in the administration of an enema or a cathartic. Many aged patients develop an atonic colon which may be filled with fecal material resulting in massive fecal impaction which can produce some degree of intestinal obstruction. In cases of this type, enemas and cathartics often relieve the situation. Consequently, three or four days may elapse after the onset of a true obstruction especially if the colon is involved before the

onset of distention and vomiting results in consultation with the surgeon

With an accurate diagnosis an adequate properly placed incision may be made and the surgical procedure carried out with a minimum of trauma. Elderly patients do not stand extensive exploratory procedures as well as younger people because their physiologic reserve is diminished. For this reason they are much less able to withstand the shock of prolonged or rough handling of tissues and excessive blood loss.

FACTORS AND RISKS ACCOMPANYING SURGERY

When surgery is indicated in the aged prolonged bed rest, sudden changes of environment and diet and abrupt curtailment of previous habits should be avoided. The maintenance of an agreeable atmosphere should be encouraged. In general elderly patients are less worried about surgery than younger individuals and are more likely to accept it with resignation. However, this resigned attitude on the part of the aged may act as a two-edged sword. Although the aged patient accepts an imminent surgical procedure with a calm and fatalistic philosophy, he may become negativistic so that the resulting lack of cooperation may have serious consequences. A confident and optimistic bedside approach on the part of the surgeon will help to give the elderly patient a more hopeful outlook. He must be made to feel that he is wanted and needed. The presence of friends and relatives during the pre operative period and their demonstration of interest in his welfare will do much to assure a successful outcome not only to the surgical procedure but also to the subsequent convalescence.

It has been demonstrated that the surgical risk in the aged is increased when operative intervention is required on hot and humid days. For this reason it is important that elderly patients undergoing major surgery be put in an atmosphere in which there is proper heat dissipation. This is particularly essential for those individuals living in temperate zones or those who are sensitive to heat and humidity. Elderly patients are susceptible to heat and are likely to suffer from heat prostration or exhaustion. This is apt to occur if the pa-

tient is heavily draped in a humid operating room and sweats losing considerable amounts of water and salt during the course of a prolonged surgical procedure. To avoid this, elective surgical procedures should be postponed from a hot day to a cooler one and if possible the surgery should be performed in an air conditioned amphitheater. In addition chloride and fluid loss following excessive perspiration should be controlled by adequate intravenous alimentation.

The best results are obtained in the surgical management of elderly patients when there is sufficient time to correct electrolyte imbalances and restore the plasma protein level to normal. In addition a vigorous attempt should be made to correct vitamin deficiency states and to hydrate the patient adequately. Many of these aged patients are debilitated and dehydrated as a result of disease processes or poor dietary habits. This is particularly true of those elderly individuals in institutions for the aged. Many elderly patients have developed water deficits because they take in little water during the course of the day. Special attention should be given to the oral hygiene of the elderly patient because bad or missing teeth or poorly fitting bridges and plates which produce pain not only have a destructive effect in preventing the patient from taking sufficient food and in limiting proper digestion but also may act as a continuous source of discomfort and irritation.

It is important that the plasma protein level be restored to normal since the protein helps to control and stabilize the distribution of body fluids. In addition proteins aid in antibody formation. A low protein level which is caused by an inadequate ingestion of protein, a diminished assimilation of protein due to an increased body breakdown or impaired synthesis decreases the patient's chances of survival. One of the manifestations of hypoproteinemia is the development of edema. A depletion of the protein reserve which is accompanied by dehydration may be masked by a high protein level in the concentrated blood. Patients admitted to the hospital with a low protein blood level become much better surgical risks if they are fasted for four or five days before surgery and the protein level is brought up to normal. It has been demonstrated that these patients can withstand

surgical procedures much better and with less danger of wound disruption and infection than can the patients suffering from nitrogen deficits. The most satisfactory method to combat hypoproteinemia is the administration of ground meat or hydrolyzed meat by mouth. In those cases in which this is not possible tube feeding after the method of Barron is desirable. Blood transfusions or plasma given intravenously are of value in such cases.

In a recent clinical study of the blood volume in geriatric surgery Behring Bosch and Carter reported that elderly patients have significantly greater blood volume deficits than younger patients. They point out that ordinary laboratory data are unreliable as indicators of circulating blood volume and that clinical judgement is often more reliable. The correction of such deficits in the elderly patient results in a significantly lowered mortality rate. Their studies indicate that blood volume repletion is not only beneficial but mandatory for patients with cardiac disease. Cardiac failure and shock are eliminated as a cause of death by pre operative blood volume replacement. Contrary to popular opinion quantitative replacement does not produce overloading or embarrassment to the cardiovascular system. As a result of the proper pre operative care and replacement of the blood volume extensive surgery in the elderly patient is not only possible but the postoperative rehabilitation is considerably smoother. Once such blood volume deficits are corrected age in itself is no contra indication to surgery.

Many of these old patients have an inefficient kidney function which results in a state of chronic dehydration. As a result serious illness such as acute intestinal obstruction may throw them rapidly into a sodium deficiency which after a few days may develop into a potassium deficiency further complicating the picture. In addition the routine blood count may be misleading because there may be a contracted vascular bed and a contracted blood volume. In any patient who has recently lost a good deal of weight one should be immediately suspicious of a decrease in circulating blood volume and a transfusion should be given before as well as during surgery.

All patients in the older age groups should be

checked carefully for the possibility of diabetes. With diabetes there is a greater incidence of degenerative disease so that infection, gangrene, poor wound healing and acidosis are apt to occur. A controlled diet and the use of insulin are indicated to help tide the patient over the surgical procedure. In such individuals the use of insulin, glucose water and saline given in proper proportions is sufficient to restore the glycogen to the liver and adequately hydrate the patient. At Grace Hospital it is our practice to turn the management of the diabetic problem over to an internist especially interested in the care of diabetics.

The problem of pulmonary disease, either acute or chronic, is very important in the aged. All elderly patients must be encouraged to get up early the morning of the operation and walk about so that they will cough thoroughly and clean out the tracheobronchial tree and thus rid it of its mucous plugs. For the same reason premedication should be very light the night before surgery and the pre operative medication should be postponed until the coughing and expectoration have been carried out. Postoperatively every effort should be made to prevent pulmonary complications. To accomplish this early ambulation is essential and the patient must be forced to move around in bed as well as out of bed. He must be told of the danger of shallow breathing and must be urged to cough up mucous plugs. During the postoperative period deep sedation is to be scrupulously avoided. The liberal use of penicillin has done much to control the harmful effects of low grade and minor infections so often associated with surgery in the aged. Many believe that the prophylactic use of penicillin 48 hours prior to geriatric surgery is indicated. In preparing such patients for surgery upon the gastro intestinal tract the elimination of pathogenic bacteria in the intestinal flora by the sulfonamides and neomycin has done much to minimize infection and peritonitis. The classic method of cleaning the gastro intestinal tract using castor oil and enemas should not be ignored but should be used in conjunction with the liberal use of the antibiotics and sulfonamides.

Just prior to surgery and during the period of time in which supportive and diagnostic studies are being carried out a long intestinal decompression

sion tube should be passed and vigorous efforts made to assure its passage as far down the gastrointestinal tract as possible. However in no event should the patient be subjected to any delay in surgical intervention because of difficulties in successful intubation. In obstructions of the right or left colon the use of the long intestinal decompression tube is of value in preventing the swelling of air and in the control of the postoperative small bowel ileus which invariably results. It must be remembered that in colonic obstructions in the aged as well as in the younger age groups the long intestinal decompression tube is simply an adjunct to the surgical procedure which decompresses or defunctionizes the small bowel proximal to the point of obstruction. In general cecostomy is used as a decompressing measure in obstructions of the right colon and transverse colostomy for obstructive lesions of the left colon.

The anesthetic risks associated with gastrointestinal surgery in the aged patient are greater than in the younger age groups. Accumulated experience has showed that the preoperative use of small doses of morphine is satisfactory to prepare the patient for surgical intervention. The safest anesthesia for use in the aged is a local anesthesia with procaine and without adrenalin. However this type of anesthesia does not permit exploratory procedures. In aged patients a diversionary procedure such as cecostomy or transverse colostomy may be safely and adequately performed under local anesthesia. When the use of general anesthesia is required the endotracheal administration is most desirable because an adequate airway is assured. In addition the tracheobronchial tree may be aspirated prior to the introduction of the endotracheal tube and the tracheobronchial tree may be aspirated of mucous plugs at the conclusion of surgery by means of a bronchoscope. A combination of cyclopropane and oxygen is widely used in such endotracheal anesthesia because of the high concentration of oxygen it has the additional advantage of wearing off rapidly thus eliminating the prolonged period of unconsciousness after the cessation of anesthesia. Ether is apt to be irritating to the respiratory tract and may predispose to the outpouring of mucus so that pulmonary complications are likely to be more

frequent. In the elderly cardiac patient however ether is the anesthetic of choice. The mixture created with nitrous oxide and oxygen may be quite dangerous for prolonged surgical procedures. This combination however is useful in short operative cases where total relaxation is not required. In those aged patients in whom a fall in blood pressure is apt to be dangerous spinal anesthesia should be avoided. In an occasional case caudal anesthesia may be the choice of the anesthesiologist. Pentothal is widely used in some hospitals for the induction of anesthesia and the introduction of the endotracheal tube. In any event the employment of a qualified medical anesthesiologist is more important than the choice of anesthetic to be used.

During surgery it must be remembered that the aged stand blood loss and oxygen lack very poorly. They may go into shock and recover from it very slowly. In addition low levels of blood pressure are much more dangerous in an elderly patient than in the young or middle aged individual. Hypotension is dangerous to the elderly individual because it may bring about coronary or cerebral thrombosis. Therefore surgery should be carried out with a minimum of blood loss and the least possible trauma to the tissues. The aged go into shock more slowly and subtly than younger patients so that the recognition of the shock state may be difficult and the patient's reaction to treatment of the hypotension is correspondingly slow. For this reason all shock producing acts such as rough handling of tissues and organs and unnecessary loss of blood and body heat must be avoided. In addition to gentle handling of the tissues and effective hemostasis adequate replacement of blood loss is of the greatest importance during the surgical procedure since the aged do not tolerate excessive blood loss.

The surgical procedure should be planned well in advance and barring unforeseen occurrence should be performed as rapidly as is consistent with the skill and dexterity of the operating surgeon. However it is better to carry out the surgical procedure slowly and meticulously than to do so too rapidly in an effort to shorten the period of anesthesia. In some instances a staged operation may be preferable to a prolonged extensive procedure. In an effort to find the point

of obstruction should be avoided. A continuous drip of intravenous whole blood or plasma should be instituted in all cases during surgery.

To some extent the choice of the specific surgical procedure to be employed in an aged individual must be made by balancing the factor of the number of years of comfortable life which one might expect the patient to enjoy if all goes well against his ability to withstand the trauma involved in the operation required to accomplish this purpose. In addition the statistical hazards of the various operative procedures as well as the skill of the operating surgeon in the performance of the procedure in question must all be considered.

The physical status of the patient at the time of the obstruction is the best guide to the ultimate prognosis. It is generally agreed that the high percentage of deaths in the older age group following surgery is in the majority of cases the result of pre-existing degenerative disease. Surgery of great magnitude may be performed upon elderly patients who although chronologically old may be physically much younger as indicated by the condition of their arteries. These individuals are often relatively free from the deteriorative processes and the diseases associated with the aged

ETIOLOGY AND TREATMENT

There are varied causes of intestinal obstruction in the aged but the list of etiologic factors is relatively small when compared with the younger age groups. The following causes are responsible for the majority of gastro-intestinal tract obstructions in the aged.

Hernia

Incarcerated hernia is the most frequent cause of acute intestinal obstruction in the elderly patient. The types of hernia responsible for this are inguinal, femoral, umbilical and incisional in that order of frequency. The inguinal hernia constitutes by far the largest group of cases. Although femoral hernia was found second in frequency to inguinal hernia, gangrene of the bowel was found to result more frequently from femoral hernia than from incarcerated inguinal hernias. In addition incarcerated femoral hernia as a cause of intestinal obstruction not infrequently escapes de-

tection unless it is specifically looked for. On a survey film it is not uncommon for the radiologic demonstration of small bowel distention indicating acute intestinal obstruction to so overshadow the presence of a small lump in the groin indicating a femoral hernia that it leads the attending surgeon into an erroneous diagnosis.

Diagnosis. The symptoms noted by patients with incarcerated hernias producing intestinal obstruction consist of pain in the region of the hernia associated with an irreducible mass. This is generally followed by colicky abdominal pain, vomiting, distention and inability to move the bowels. In many cases the onset of symptoms is so acute and the pain so severe that early surgical consultation is sought. In most cases immediate surgery follows. The presence of a recognizable hernia which proves to be irreducible is so clear-cut that an accurate diagnosis is possible and the indications for immediate surgery incontrovertible. In some cases however a diagnosis may be difficult. This is particularly true when a Richter's hernia is present or when a small femoral hernia is the cause of the obstruction. Here the diagnostic difficulty is largely due to the failure of many surgeons to recognize the presence of these hernias because the obstruction may not be complete, only a small portion of the lumen of the bowel being strangulated within the hernial sac and no interruption to the continuity of the small bowel being present.

Treatment. In all cases of hernia an opportunity to correct underlying chronic disease or to improve the condition of the patient may not be available because of the urgency of the surgical procedure. However during the interval of time required to prepare the room for surgery the patient may be hydrated. If there is a definite anemia a transfusion may be given and may be repeated on the table particularly if extensive dissection is required. Antibiotics may be given immediately upon admission and a long intestinal decompression tube may be passed. The correction of blood volume deficits by transfusion is an important feature of the pre-operative as well as the operative and postoperative care of the patient. In all such cases surgery should consist of the simplest possible procedure. Freeing of the ob-

structed loop with reduction of the hernia or repair are desirable whenever possible. In no case should the bowel be permitted to fall back into the peritoneal cavity without being carefully examined for points of strangulation. Femoral hernia is particularly likely to strangulate and early necrosis is not uncommon. Most of the deaths reported as a result of intestinal obstruction due to hernia have been from this source. Once the hernial sac has been opened, its contents carefully examined and it has been determined that strangulation is not present, great care should be taken in returning the bowel to the peritoneal cavity. Any bowel whose viability is questionable should not be returned. The return of such compromised bowel to the peritoneal cavity may result in subsequent gangrenous changes and perforation or the later development of a stenotic area with intestinal obstruction. All such questionable bowel should be resected at the time of surgery before being replaced in the peritoneal cavity.

A particularly dangerous procedure is the reduction of a recurrent inguinal hernia en masse. In such a case the bowel may remain obstructed within the peritoneal cavity and subsequently may undergo necrosis with perforation. Because strangulation of the bowel is present in an appreciable percentage of femoral hernias associated with intestinal obstruction, some surgeons suggest that a transabdominal approach be used in such cases. This technic permits an easy resection of the strangulated bowel, a procedure which might be difficult through the orthodox incision for the treatment of a femoral hernia with incarceration.

In those cases in which hernia is the cause of intestinal obstruction, it has been found that the duration of the obstructive process resulting from the hernia is an extremely important factor in determining the morbidity and ultimate mortality. This factor is more important than the age of the patient or his coexisting physical defects. Any delay in surgical intervention for the purpose of attempting decompression by the long intestinal decompression tube is dangerous.

Neoplasms

The second most common cause of intestinal obstruction in the aged is tumor. Of these carci-

noma constitutes by far the largest group. The sigmoid colon is the most common site for the obstructive process caused by such tumors with the rectum, descending colon and the hepatic flexure coming next in that order of frequency. Patients presenting tumors of the colon with intestinal obstruction are generally brought to the surgeon at a much later date than those with small bowel tumors causing obstruction. This is due to a failure on the part of the attending physician to recognize the fact that an obstructive mechanism is present.

Diagnosis. Many of these elderly patients are chronically constipated and present an atonic colon with impacted feces in the rectum. Many of them give a history of constipation for three or four days with relief by enemas. Even the association of abdominal pain with a failure to move the bowel for two or three days is not considered too significant. In these cases it is only after the onset of distention and vomiting that these individuals are admitted to the hospital and the possibility of intestinal obstruction is considered. Because the symptoms are insidious in onset and only slowly progressive, the diagnosis may be delayed. A history of a slowly progressive lesion when associated with a history of constipation, blood in the stool and a loss of weight is highly significant. Many cases may be readily diagnosed by rectal or sigmoidoscopic examination. In those cases in which the obstructing process is beyond the range of the sigmoidoscope—25 cm—a barium enema will serve to make an exact diagnosis as well as to localize the lesion.

Treatment. In all cases of this type the preoperative treatment consists of the correction of anemia, electrolyte imbalance and protein deficiency and the administration of blood. In the presence of acute colonic obstruction a defunctionizing colostomy is the procedure of choice. This is to be followed at a later date by resection of the obstructed bowel and end to end anastomosis.

In those cases in which the obstructive process is in the right colon, a preliminary exteriorization cecostomy is the procedure of choice except when the obstructing lesion is at the ileocecal valve. When this occurs proximal decompression may be obtained by the use of the long intestinal de-

compression tube or a diversionary ileotransverse colostomy to be followed at a later date by a resection of the right colon.

In those cases in which the obstructing process is not complete and in which following an enema and removal of fecal impaction the bowels may move spontaneously a period of pre-operative management may be used so that a primary resection and anastomosis can be performed. Although a long intestinal decompression tube should be used for the purpose of decompressing the small bowel needless delay in order to assure the successful passage of this tube before the performance of cecostomy or colostomy as a primary decompressive measure should not be permitted. This is especially true of those cases in which the ileocecal valve is competent. In this event immediate decompression of the colon is indicated. The use of the long intestinal decompression tube in an effort to decompress obstructions of the colon is an extremely dangerous procedure—particularly in the case of obstructions involving the left colon either with or without a competent ileocecal valve. In those cases in which the valve is competent cecal blowout is an imminent danger whereas in those cases in which the ileocecal valve is incompetent perforation at the site of the obstructing malignancy may occur. It has been observed on occasion that the colostomy or cecostomy may fail to function for the first three to four days after operation as a result of small bowel ileus incidental to the surgical procedure. It is for this reason that the long intestinal decompression tube should be used to tide the patient over the first few days until peristaltic activity becomes reestablished. When this occurs the colostomy or cecostomy begins to function properly. It has been repeatedly pointed out that the prolonged use of the long intestinal decompression tube in the aged not only does not better the patient's chances of survival but may even decrease his chances if the colon is not successfully decompressed.

Adhesions

Adhesions rank third as a cause of intestinal obstruction in the aged. Most of these patients give a history of previous surgery which permits a diagnosis to be made with ease. In most of the

cases reported operative procedures on the appendix and pelvic organs were found to have been predisposing factors in the development of such adhesions. Diverticulitis protrusions of the small bowel through defects in the omentum, internal hernia, acute pancreatitis, the conservative management of perforated ulcers, and the organization of hemorrhage within the peritoneal cavity due to trauma are among the nonoperative causes of adhesions.

Diagnosis. Such small bowel obstructions due to adhesions are usually characterized by a sudden and acute onset of symptoms with severe cramping pain. Vomiting, constipation and intestinal distention are early and prominent features. These patients are very sick and medical attention is sought early. In most cases the signs and symptoms of small bowel obstruction are obvious and the presence of abdominal scars is helpful in arriving at a correct diagnosis when correlated with the radiologic findings of small bowel gaseous distention with little or no gas in the colon.

Treatment. Early operation is essential after correction of electrolyte imbalance, proper hydration and treatment of the intestinal distention by passage of a long intestinal decompression tube. It has been found that prolonged and ineffectual attempts at intubation are associated with a high mortality in patients in this age group. The association of small bowel obstruction with large bowel obstruction must be kept in mind at the time of surgery. For this reason adequate exploration should be used and a survey of the small bowel as well as the colon should be made in all cases. It has been emphasized that in the management of small bowel obstructions in the aged early surgical intervention and a minimum surgical procedure improve the chances of survival.

Foreign Bodies

Foreign bodies in the gastrointestinal tract are not uncommon as a cause of intestinal obstruction in the aged. The most common of these foreign bodies are gallstones with peach pits, meat bones, and in an occasional case a long intestinal tube also found as the obstructing mechanism.

Diagnosis. These patients with acute small bowel obstruction caused by foreign bodies may

present typical clinical and radiologic evidence of acute small bowel obstruction. On the other hand the onset may be so insidious and the clinical picture so confused that a correct diagnosis may not be made until relatively late in the course of the disease. This is especially true in cases in which gallstones produce obstruction. Whereas in former years a correct diagnosis of this type of obstruction was rarely made prior to surgery, in recent years as a result of the increased attention given to this lesion by radiologists a correct diagnosis is being made in an increasingly high percentage of cases. In a small percentage of cases the radiologist may demonstrate a radiopaque gallstone in the distended small bowel. Clinical evidence of intestinal obstruction, radiologic evidence of small bowel distention without gas in the colon, and air filled bile radicles constitute diagnostic criteria for gallstone obstruction even if the stone itself is not demonstrable.

Treatment. In the surgical treatment of lesions of this type whenever possible one should not open the bowel to correct the obstruction. When the foreign body can be milked through the ileocecal valve it is almost invariably excreted per rectum except when there is colonic obstruction. When the necessity for opening the small bowel does occur the defensive mechanisms of these elderly patients should be bolstered in all possible ways since an increase in morbidity and mortality is to be expected.

Volvulus

One of the most serious types of obstruction found in the aged is volvulus of the sigmoid colon. In these cases strangulation occurs relatively early and rapidly leads to gangrene. In cases of this type unless surgical relief is undertaken early the results are generally poor. Simple detorsion is the procedure of choice unless gangrene of the bowel has occurred. In such cases primary resection and anastomosis are desirable for the good risk patients and simple detorsion and exteriorization for the poor risk patient. The exteriorized necrotic loop is cut away after application of occluding clamps. These are removed in two to three days and a double barrelled colostomy permitted to form. Secondary closure of the colostomy is

deferred until the condition of the patient is favorable.

POSTOPERATIVE TREATMENT

Experience has shown that the high mortality associated with intestinal obstruction in the aged is fundamentally the result of the obstructive process itself and its treatment rather than the patient's age. The most important factor is the duration of obstruction. The longer the obstruction exists prior to surgery the poorer are the chances of recovery. Because in many of these elderly patients a diagnosis of obstruction is not made until relatively late the results in general are not too good. Emphasis must be placed upon earlier diagnosis and earlier treatment in order to obtain the lowest possible mortality rate. A reluctance on the part of the surgeon to operate upon these old patients or prolonged attempts at intubation only increase the delay in adequate surgical therapy and proportionately decrease the patient's chances for survival. The very best results in the surgical management of intestinal obstruction in the aged occur when an operation of the simplest possible type is performed at the earliest possible moment. At the time of surgery when bowel resection is required a correction of incidental pathology should not be undertaken.

Postoperative pulmonary lesions such as atelectasis, massive collapse of the lung, and pneumonia are important contributing factors increasing the mortality rate in this age group. These pulmonary complications are the result of an interference with the respiratory movements and an inability on the part of the patient to evacuate bronchial secretions during as well as after surgery. After operation but before the patient leaves the operating room aspiration of the tracheobronchial tree by the anesthetist using a bronchoscope is a highly desirable procedure and will do much to decrease these pulmonary complications. The aspiration of thickened bronchial mucous plugs or aspirated food particles brought up by the patient during the time he can be extremely dangerous. To prevent the aspiration of food particles the stomach should be emptied prior to surgery by means of the Levine tube or an intestinal decompression tube. As an additional measure to prevent pulmonary complica-

eration the patients should be ambulated early. Early ambulation in the aged means walking as soon as they have recovered from the anesthesia usually within six hours from the time of surgery. From this time on the patient must be encouraged to remain ambulatory as much as possible. The only contra-indication to early ambulation in the aged patient occurs in those individuals who are in danger of cardiac failure who have profound shock or who have suffered from severe hemorrhage.

Oxygen therapy should be routinely used the first 24 hours after operation. Oxygenation in the aged especially those who have had surgical procedures in the upper abdomen is of great value. As a result of the increased oxygenation the pulmonary and tissue respiration are increased. The vital capacity is increased and the cardiac load is diminished. It has been estimated that the administration of 100 per cent oxygen causes a 10 to 15 per cent increase in oxygen in the arterial blood and a slightly higher increase in the venous blood. This can do much to reduce the mortality from shock, acute cardiac decompensation, circulatory failure and pulmonary edema as well as abdominal distention. In those cases in which the use of the oxygen tent is not practical nasal cannulas may be inserted.

In those patients in whom there is a history of previous phlebitis or when varicosities exist bandaging of the legs with Ace bandages and early ambulation are of the greatest importance. Early ambulation has reduced to some small extent the incidence of phlebothrombosis, thrombophlebitis, pulmonary infarction and fatal embolus. In any case in which such complications appear the use of anticoagulants should be resorted to at the earliest possible moment. The highest incidence of thrombosis and embolism in the older age group is apt to occur during the winter months, the major etiologic factor in such cases being stasis in the deep leg veins. Daily examination of the legs for signs of phlebothrombosis or thrombophlebitis is essential. A positive Homans sign suggests the presence of phlebothrombosis particularly if the temperature pulse and respirations are rising. Although early ambulation has done much to improve the physiologic mechanisms of the patient

it has not completely eliminated the hazards of embolus due to phlebothrombosis.

In surgery upon the elderly patient great care must be exercised to avoid the possible development of decubitus ulcers. These are often multiple and in the aged surgical patient generally occur over the sacral region and the trochanters. The presence of such open infected wounds may begin a serious downhill course and may result in a fatality in an otherwise successfully operated patient. Early ambulation has done much to prevent the development of such lesions.

In the elderly patient the proper maintenance of a fluid and electrolyte balance in the postoperative period must be tempered with considerable judgement. One must consider the myocardial reserve and the ability of the patient to utilize the infusions of fluid given intravenously over a short period of time. The urinary volume of the aged patient should be at least 1000 cc daily and an additional 1000 cc must be given to replace the fluid lost through sweating and breathing. All intravenous fluids given to the aged patient must be administered very slowly to avoid the possible danger of overloading the circulation. Urine with a specific gravity below 1.015 is inadequate for proper hydration and nitrogen excretion by the kidneys. Imbalance of physiochemical processes is rather common after surgery in the aged. Anemia, dehydration, electrolyte imbalance, hypoproteinemia and vitamin deficiencies are particularly apt to occur in this age group and are particularly common in those individuals presenting malignant disease or pyloric obstruction in addition to intestinal obstruction. Blood studies such as nonprotein nitrogen, carbon dioxide combining power, chlorides, sodium and potassium should be done not only pre-operatively but at frequent intervals postoperatively in order to be sure that proper hydration is being maintained and that the electrolytes are in normal balance.

Great attention should be given to the cardiac status of the patient. Ventricular fibrillation and cardiac irregularities are not uncommon following prolonged anesthesia. Cardiac arrest may occur as a result of vagal stimulation and is particularly apt to occur as a result of surgery in the aged.

patient who is somewhat apprehensive and who during the course of the surgical procedure has become anxious. It is extremely important that such periods of anxiety be avoided by adequate oxygen and a free airway at all times.

Since elderly patients are apt to develop fecal impactions, careful attention should be given to any complaints of fullness in the perineum or rectum. Unsatisfying frequent small liquid bowel movements should immediately call for an examination of the rectum.

It has been pointed out frequently by surgeons experienced in geriatric surgery that one of the most important factors in the management of the elderly postoperative patient is the protection from emotional upset. Old patients generally frighten

easily and become despondent. For this reason the surgeon must always present an optimistic outlook. He should point to some favorable development of the case each day and should avoid detail of the surgical procedure. The patient must be made to feel that everyone is interested in his welfare and desires him to get well. The habits of a lifetime with regard to smoking and the intake of alcohol should not be broken during the postoperative period. Whenever possible the patient should be permitted considerable latitude in these two particulars. It has been clearly demonstrated that scrupulous attention to the details of these special problems accompanying surgery is highly rewarded by a low mortality rate in this age group for which results in the past were so poor.

RADIOLOGIC DIAGNOSIS OF GASTRO- INTESTINAL OBSTRUCTION

The diagnostic significance of intestinal gas shadows in suspected cases of intestinal obstruction was described in 1911 by Schwarz. The first American to point out the value of radiologic examination and the importance of the intestinal gas shadows in cases of intestinal obstruction was Case. Case's work appeared in 1914 and throughout the years he has been a very strong proponent of early radiologic study. Klobner showed that the radiologic diagnosis of intestinal distention without the use of barium was not only feasible but also quite accurate. As a result a survey film of the abdomen without the use of a contrast medium has become an established procedure in all cases of suspected intestinal obstruction. It has been demonstrated that the development of intestinal gas precedes the fluid level in the radiologic examination. Ochsner demonstrated in the early 1930's that intestinal gas within the small bowel could be found on a survey film of the abdomen within one hour from the time of onset in cases of strangulating obstructions and within four hours in nonstrangulating obstruction.

The technic of radiologic examination is of the greatest importance in arriving at a correct diagnosis. If the patient maintains the erect position before the survey film is taken, fluid levels may be demonstrated at a rather early date. In determining the extent of intestinal gas, the position generally assumed by the patient is the prone position which sharply outlines the bowel; however, this position disturbs the relationship of the loops of distended bowel as a result of pressure. The oblique position and the erect position are used to demonstrate fluid levels.

The radiologic criterion used to diagnose small bowel obstruction is the presence of a small bowel distention with gas in the absence of evident gas in the colon. This radiographic demonstration is considered to be pathognomonic of small bowel obstruction, providing the colon has not been emptied by previous enemas. It should be noted, however, that the presence of intestinal gas is a normal



FIG 232 Jejunal obstruction. Upright film showing fluid levels.



FIG 233 Small bowel obstruction due to adhesions. Note the small bowel distention with little or no gas in the colon. This is radiologic evidence of small bowel obstruction.

finding in children under the age of two. Thus gas leaves the small bowel very rapidly thereafter so that at the age of 10 little or no gas is found in the small intestine.

A history of the injection of morphine is important in all cases in which a survey film of the abdomen is taken for diagnostic purposes. It is well known that appreciable amounts of intestinal gas may appear in the small and large intestine if the patient has been injected with morphine.

The radiologic signs of small bowel obstruction depend upon the presence of gas within the lumen of the bowel. Any one or all of the following findings are highly suggestive of small bowel obstruction:

- 1 The presence of fluid levels in the small bowel.
- 2 A stepladder arrangement of loops of bowel.
- 3 The absence of gas in the colon.
- 4 Those cases in which gas is found both in

the large and small bowel suggest a paralytic ileus or that the obstruction is incomplete and that gas has gone through the obstructing process and into the colon. Another possibility in those cases in which both the small and large bowel are distended with gas is the presence of a large bowel obstruction as well as a simultaneous small bowel obstruction due to a loop of intestine becoming adherent to an obstructive carcinoma of the colon. This occurrence is by no means rare.

In general the visible distention of isolated loops of small bowel without colonic distention is usually considered indicative of mechanical small bowel obstruction which may require surgical intervention. However the radiologic findings must be correlated with the clinical findings since many errors in diagnosis are possible if this correlation is not made. A variety of clinical entities may be associated with marked small bowel distention without any intestinal obstruction being present.



FIG 234 Notice the use of intestinal gas as a contrast medium. The stomach visualizes well as do the pylorus and the duodenal cap.

Among these are ureteral calculus producing a reflex paralytic ileus, poliomyelitis, tubes dorsalis and diabetic acidosis. On the other hand it must also be remembered that a paralytic ileus may be associated with a mechanical small bowel obstruction and as a result the true picture of a mechanical obstruction may be masked by the radiologic findings indicative of a paralytic ileus. For these reasons the intestinal gas pattern noted on the survey film must be used as a diagnostic procedure and correlated with the clinical findings as well as the laboratory findings in order to avoid a serious diagnostic error. To draw conclusions as to the presence or absence of intestinal obstruction solely by an examination of the survey film alone is to invite disaster. It has been amply demonstrated not only that a negative report from the radiologist after an examination of the survey film does not rule out the possibility that intestinal obstruction may be present but also that a positive report of mechanical intestinal obstruction when not correlated with the clinical findings is no proof that such an obstruction is present. In addition the survey film should not be used to diagnose early intestinal strangulation. In late intestinal strangulation however the radiologic findings are quite characteristic.

In those cases in which the radiologist confirms our clinical findings, one may consider the diagnosis to be beyond much reasonable doubt. When the radiologic findings are positive but a clinical diagnosis is lacking, the surgeon had best review his position to determine whether a true obstruction is present which is not manifested clinically or whether the radiologic findings are misleading. In the face of absolute clinical findings of intestinal obstruction and a radiologic diagnosis of a normal gastro intestinal tract without evidence of intestinal obstruction the surgeon should ignore the radiologic findings and follow his clinical judgment. In any event close cooperation between the radiologist and surgeon is highly desirable if errors of commission and omission are to be avoided.

RADIOLOGIC DIAGNOSIS VIA GAS PATTERNS

Technic

The technic used to examine any patient suspected of having intestinal obstruction must be

varied to suit the specific problem presented. From the radiologic point of view there are four recognizable types of patients:

- 1 In this group are those patients in whom there is positive clinical evidence of intestinal obstruction. In this group the radiologic findings are not absolutely essential to an accurate diagnosis but are important factors in determining the exact location of the obstructive process and at times the degree of obstruction. These patients usually present the most advanced type of bowel obstruction. A survey film of the abdomen will often give valuable information as to the presence of large or small bowel obstruction and in the case of a large bowel obstruction will often reveal the site of the obstructive process or its type.
- 2 This group is composed of those patients in whom there is no clinical evidence of obstruction or if clinical evidence is present it is only suggestive of partial obstruction. The radiologist can be of inestimable value for this group since he offers the only means of establishing a positive diagnosis.
- 3 In this group are those individuals in whom there is clinical evidence of obstruction but for whom the radiologic findings are negative. In this instance the clinical judgement of the attending physician is of the greatest importance. It is essential however that the radiologist and surgeon collaborate closely before surgical intervention is undertaken on patients of this type. These patients require repeated radiologic examination over a period of three or four hours before a final decision can be made.
- 4 This group is composed of those patients in whom there is both radiologic and clinical evidence of obstruction and yet at operation the surgeon may fail to find the obstructive process. The only reasonable explanation for this occurrence is that the obstructing process released itself either spontaneously or as a result of anesthesia prior to surgery. Cases of this type which are rare are either internal herniations which reduce themselves spontaneously or small mesenteric emboli.

It is generally desirable for the patient to be brought to the X ray department where the Bucky diaphragm can be used for sharp definition. In those cases where this is not possible satisfactory films may be obtained with a portable X ray at the patient's bedside. The first film is taken with the patient in the supine position. Although this position does not show fluid levels, the film will give general information as to the degree of distention and may be sufficient to establish the diagnosis. If it is at all possible the second film should be taken with the patient in the erect position or sitting up. The erect film may be taken with the patient standing or secured on a tilting table. If the patient's condition does not permit this film may be taken with the patient sitting upright. A third film is then made with the patient lying horizontally first on one side and then on the other. This will show the movement of gas and fluid intestinal gas patterns and fluid levels. Free fluid

in the peritoneal cavity as well as the finding of fluid and gas within the bowel can be obtained with a horizontal exposure with the patient lying on one side and then on the other. In order to study the peritoneal fat line it is essential that soft tissue detail is obtainable. For this purpose films should be underexposed if the peritoneal fat line is to be used as a diagnostic point.

A 14 by 17 film should be used in all cases that the entire abdominal cavity is included in the film. Since the diaphragm is elevated as a result of the intestinal distention the film should be centered on a point higher than would be normal. This will assure a clear view of the splenic flexure. No enemata should be given before taking film. Whenever possible the radiologic examination of the patient should begin with an X ray of the chest. This often gives one a clue as to the etiology of the obstruction. Metastatic carcinoma of the lung, tuberculosis, enlargement of the left ventricle and other pulmonary diseases may indicate the primary source from which emboli or metastases are disseminated.

Spencer and Thraxter suggest that in any examination of the survey film the following points should be kept in mind:

1. Abnormal gas shadows due to gas within the bowel, a pneumoperitoneum and air in the biliary tract.
2. Possible cause of mechanical or reflex distention such as gallstones, renal stones, fecaliths and foreign bodies.
3. Abnormal soft tissue shadows.
4. Bone abnormalities.

Diagnosis

It has been amply demonstrated that gas is a contrast medium which will outline the gastrointestinal tract. It is the accumulation of the gas in the lumen of the gastrointestinal tract which produces the radiologic evidence that permits a diagnosis of intestinal obstruction. The ascension of fluid with this gas is confirmatory evidence. In any case in which there is a delay or an impairment in the propulsion of the intestinal content from the mouth to the anal outlet, stagnation of intestinal gas and fluid will occur which so distends the bowel above the point of obstruction that



Fig. 23. X ray taken with a bedside unit. Intestinal distention is present but notice the poor detail shown on the portable film. For this reason the Bucky should be used and the patient taken to X ray whenever possible.

it can be seen radiologically with the proper technique. In the early stages of the process the distending element is gas and in the later stages it is a mixture of gas and fluid. The appearance of the obstructed bowel on a survey film depends upon the degree of obstruction as well as upon its location. If the jejunal loops become distended a typical herringbone appearance may be found due to the stretching of the circular folds of the bowel or the valvulae conniventes. These valvulae conniventes are reduplications of the mucous membrane. Since the two layers of the fold are bound together by submucous tissue the folds are not obliterated by intestinal distention in the upper jejunum and as a result they give rise to the typical herringbone pattern. These folds tend to disappear however as one proceeds down the gastrointestinal tract so that they do not appear in the terminal ileum. Because the valvulae conniventes encircle the intestine at right angles to its long axis when the bowel is distended and the jejunum and upper ileum lie in a horizontal direction one superimposed upon the other this gives rise to a typical stepladder appearance with a herringbone pattern. In the ileum because of the absence of valvulae conniventes distention of the ileal wall will produce a smooth appearance in contradistinction to the herringbone pattern of jejunal obstruction. The distended loops of bowel may not lie one upon the other in a parallel direction but may take a vertical direction. In addition the distended smooth walled vertical loops of ileum may square off in such a fashion that they may be confused with distended loops of colon. Distention of the colon especially the left colon tends to present haustral markings. These haustral markings may be confused radiologically with the squarings which may be noted in obstructions of the terminal ileum. A diagnostic feature which is helpful in distinguishing between the intestinal gas pattern of colonic obstruction and that found in obstruction of the terminal ileum is the fact that the intestinal gas pattern of colonic obstruction generally occurs in the flanks. The ascending colon appears in the right flank and the descending colon in the left flank.

One of the most important features of the radiologic examination in those cases of intestinal

obstruction where gas is used as a contrast medium is the finding with frequent films that there is a change. The characteristic pattern may not be present simply because the intestinal gas and fluid shift. Therefore if the radiologic evidence is not confirmatory and the clinical evidence is suggestive of intestinal obstruction films taken at half hour and hourly intervals can be of great value in distinguishing the nature of the process in the bowel.

The ileocecal valve is of the greatest importance in the radiologic diagnosis of intestinal obstruction when intestinal gas is used as a contrast medium. A competent ileocecal valve will result in a markedly distended cecum and colon particularly the right colon. An incompetent ileocecal valve results in colonic distention of a far lesser degree with slowly but progressively increasing intestinal distention in which the small bowel participates. Acute colonic intestinal obstruction may result in a marked distention of the entire colon.

The presence of air under the diaphragm following abdominal surgery is an expected finding and normally lasts anywhere from 5 to 10 days when it begins to disappear as the result of absorption. This should be remembered since in any patient developing signs of intestinal obstruction after a laparotomy such accumulations of air under the diaphragm must be critically evaluated. They must not be taken to mean a pneumoperitoneum as a result of perforation of the bowel. In those cases in which previous abdominal surgery has not been performed the presence of such pneumoperitoneum indicates a coexisting perforation of the intestinal tract. Gas free in the peritoneal cavity will rise to be concentrated at the highest possible level. When the patient assumes the erect position the gas will be found under one of the domes of the diaphragm. In the lateral position it may best be seen between the right border of the liver and the abdominal wall. The dense mass of the liver enhances the contrast. Such free gas demonstrated in the peritoneal cavity of any patient not recently operated upon must be considered as a certain sign of intra abdominal perforation. However perforation of considerable magnitude may be necessary for this sign to be very well developed and moreover this sign may not be evident in every case of perforation.

Free fluid or exudate may appear in the peritoneal cavity if the process is caused by peritoneal irritation or peritonitis. This gives rise to increased density between the loops of bowel. In such cases the properitoneal fat line becomes either very sharp or completely absent. The properitoneal fat line, which varies in thickness in different individuals depending upon the amount of fat present, can generally be seen radiologically as a dark stripe in the flanks next to the bowel. When there is an accumulation of fluid within the peritoneal cavity, there is an edema of this fatty layer. This brings the absorptive capacity of the X-ray of the fat layer up to the neighboring muscle, and the contrasting dark stripe is then lost. This results in a homogenous shadow in the flank. However, in those cases in which there is marked intestinal distention associated with fluid, there may be such compression of this properitoneal fat line that it stands out as a sharp line. (Great care must be exercised in evaluating the presence of or the changes in this fat line. Although the presence of a fat line does not exclude peritonitis nor does its absence indicate fluid, the changes described previously should be watched for and evaluated.)

In the normal gastro intestinal tract the stomach almost always contains gas. This stomach gas is readily noted as a bubble beneath the left dome of the diaphragm in the fundus of the stomach. In the supine position it corresponds more or less to the shape and form of the stomach. The small bowel also contains gas, but this gas is so mixed with intestinal contents that it does not cause a shadow radiologically. The colon almost always contains gas which varies in amount with the patient and the diet. Gas may accumulate in the colon with great rapidity. Its chief source has been demonstrated to be swallowed air.

Mechanical Nonstrangulating Small Bowel Obstruction. It is generally felt that a diagnosis of complete small bowel obstruction may be made radiologically when the small bowel shows evidence of marked distention with little or no gas demonstrable in the colon. Wengenstein and Lynch demonstrated experimentally that complete obstruction of the gastro intestinal tract is followed in four to five hours by radiologic evidence of intestinal distention proximal to the point of



FIG. 236 Notice the gas filled stomach as shown by a survey film of the abdomen.

obstruction. Irimann Dahl is of the opinion that in three hours there is radiologically demonstrable intestinal distention in the obstructed patient. Despite this, he believes that it usually takes a longer period of time for the signs of intestinal distention to be sufficiently developed so that a radiologic diagnosis may be made with absolute certainty. Six or seven hours is usually required before such roentgen diagnosis is possible. In those instances in which the obstruction is complete, little or no gas passes from the ileum into the colon. In an occasional case in which the small bowel is obstructed, a small amount of gas may be found in the colon, either because the obstruction is incomplete or because large amounts of gas previously present in the colon have not as yet been completely reabsorbed or evacuated.

Competent radiologists generally can diagnose a mechanical obstruction of the small bowel from a survey film. The loops of obstructed small bowel are large and dark. The valvulae conniventes are prominent, giving an impression of barium bismuth pattern. There is a marked contrast between the



FIG. 237 Note the gas bubbles in the small bowel of a patient taking morphine. These bubbles are in isolated areas and do not indicate intestinal obstruction. This is a lateral view.



FIG. 238 Same patient as in Figure 237. Note isolated gas bubbles in the small bowel. There is no obstruction.

gas distended bowel and the rest of the tract. As a result of hyperperistalsis some of the loops of small bowel may be indistinct in contradistinction to those cases of paralytic ileus in which there is little or no motion of the small bowel with the result that until they become filled with fluid the intestinal loops are quite distinct. In mechanical obstruction the intestinal distention involves only that part of the bowel proximal to the site of obstruction. The loop of bowel distal to the point of obstruction are collapsed and empty and contain little or no gas. At the obstructive process continues however fluid may accumulate in the gastro intestinal tract which has become distended. In which cases the gas tends to collect in the upper loops while the fluid collects in the lower loops which generally gravitate into the pelvis as a result of the increase in weight.

It is often possible to differentiate nonstrangulating obstruction from strangulating obstruction radiologically. Obstruction of a nonstrangulating type presents the following characteristics:

1. Gas or fluid distended loops of small bowel lying in every conceivable plane.
2. Gas distended loops of small bowel presenting a herringbone pattern.
3. Gas distended loops of small bowel lying one loop upon another and assuming a step ladder appearance.
4. The association of gas distended loops of small bowel with little or no gas in the colon.
5. If gas is present in the colon it diminishes with repeated X rays. Increasing amounts of colonic gas are suggestive of incomplete obstruction and improvement.
6. There is little deformity of other organs in acute intestinal obstruction.

Although the degree of obstruction may be suggested by the degree of intestinal distention as



FIG 239 Same patient as in Figures 237 and 238 Barium enema showed no obstruction Upper GI series with Cantor tube in place showed no obstruction thus proving that the accumulations of gas in the small bowel were the result of morphine and were not obstructive in origin

shown radiologically this is not always true. Marked intestinal distention may be found even when the obstruction is incomplete.

Strangulating Small Bowel Obstruction

With a true strangulating obstruction the radiologic appearance may present such scanty signs that the obstruction is entirely overlooked. In such cases it is only the correlation of the clinical features of the disease with the radiologic appearance which permits an accurate diagnosis. Wangenstein attempted to determine the reliability of the appearance of intestinal gas shadows in cases associated with interference with the portal or mesenteric veins as well as in cases of peritonitis. It was found that although gas shadows in the small intestine were occasionally seen after ligation of the mesenteric or portal veins, visible gas in the small intestine did not regularly appear in the

early stages. In peritonitis and in simple obstruction however gas shadows appeared in the small bowel relatively early in the course of the disease. Thus it is important to realize that in early strangulating obstruction repeated X rays at hourly intervals may be necessary before a specific diagnosis can be made. It is the change in intestinal gas patterns and the increase or decrease in the quantity of gas found as well as the distention of the bowel which determine whether a strangulating obstruction is present. In the usual variety of strangulating obstruction one may find that an obstruction to the continuity of the small bowel produces an increase in peristaltic activity associated with severe pain. Fluid and gas accumulate in the small bowel above the point of obstruction. If the lumen at the other end of the incarcerated loop is only partially obstructed gas enters the loop but none may be able to leave it. There may be immediate spasm of the bowel if arterial occlusion has occurred. When this happens the loop of bowel involved may contain only a small amount of gas or no gas whatsoever. Thus the presence of a moderate amount of gas or the presence of fluid plus a moderate amount of gas within a fixed loop is highly suggestive of an incomplete obstruction of the strangulating type.

Mellins and Rigler in their study of the radiologic findings in cases of strangulating obstruction concluded that it was possible by a survey film alone to diagnose a strangulating type of obstruction with some degree of accuracy. The points stressed by Mellins and Rigler in the diagnosis of strangulating obstructions on a survey film are as follows:

- 1 The radiologic diagnosis of strangulating obstruction may be missed if the entity is not specifically looked for.
- 2 Strangulating obstruction is produced by a fairly constant anatomic derangement and the incarceration or twisted intestinal loop can be recognized.
- 3 There are two types of incarcerated loop. If the loop is partially closed it will be gas filled or gas and fluid filled. In the horizontal film gas will be seen in the two distended limbs of the incarcerated loop. The gas shadows will be separated by the opposed

intestinal walls which are usually edematous. As a result a thicker shadow than normal is produced. This has been called the "coffee bean" sign. If the incarcerated loop is completely closed, it will contain little or no gas. In the presence of a completely closed loop, the intestinal gas which is swallowed and distends the gastro-intestinal tract will not distend the obstructed loop which is closed at both ends. This accounts for the largest source of intestinal gas which would be excluded from the incarcerated intestinal loop. This type of obstructed loop will contain for the most part only a bloody transudate which is the result of strangulation. A small amount of swallowed air may be found in a completely closed loop if the process of closure develops over a reasonably long period of time beginning with a partial obstruction. In such cases a survey film of the abdomen will demonstrate the fluid-filled closed loop as a somewhat rounded or oval shaped soft tissue density. This gives rise to the "pseudo tumor" sign. Although the "coffee bean" sign is harder to distinguish when there is gas in the superior segment of the small bowel, the "pseudo tumor" sign is enhanced by the presence of gas in the upper regions of the small intestine.

4. Fixation of the involved loop is another of the signs usually observed. In order to demonstrate this sign, survey films should be made with the patient in the erect, the supine and the lateral decubitus positions. A failure of the intestinal loop to move is generally indicative of a closed loop strangulating type of obstruction. Short incarcerated loops will show a higher degree of fixation than longer loops which have a greater mobility.
5. A smooth or formless bowel lumen develops which can be demonstrated either within the gas-filled incarcerated loop or within the bowel just above, providing there has been sufficient distention to compromise the intramural circulation.

Although all these signs are highly suggestive of a strangulating intestinal obstruction, Wangenstein has emphasized that strangulating obstruc-

tion may occur without any evidence of abdominal distention. It has been found that distention was present in 80 per cent of nonstrangulating obstructions and in 90 per cent of early strangulating obstructions, but was found in only 40 per cent of later strangulating obstructions. Consequently, one would find on a survey film that there may be an absence or scarcity of small bowel intestinal gas shadow above the proximal end of a strangulated loop. Chesterman and Sheehan demonstrated under direct vision that mild venous obstruction produced effects that differed little from those of nonstrangulating obstruction. Severe venous obstruction on the other hand, if associated with arterial obstruction, produced marked alteration in the motor function of the rest of the small bowel. There was not only immediate spasm of the strangulated loop but also of the bowel above and below it. In fact, the effect might be transmitted to the entire small intestine and even to the pylorus, although the effect on the stomach was variable. After a period of time which varied from a few minutes to several hours, the spasm decreased and intestinal movements began. In spite of the prolonged vigorous contractions, extensive distention did not appear until retrograde venous thrombosis had occurred or peritonitis developed.

As a result of his experiments, Chesterman believed that retrograde venous thrombosis was the primary cause of the marked bowel distention regardless of whether the thrombosis was proximal or distal to an acutely strangulated loop of small intestine. If the process of retrograde thrombosis extended further on the distal side, dilatation of the bowel was found distal as well as proximal to the strangulation. These findings were confirmed not only by strangulating a loop of bowel but also by damaging the veins away from the strangulated loop. The bowel in these cases contained fluid and little or no gas. In such cases, a survey film of the abdomen is very difficult to evaluate. Survey films showing few or no signs of mechanical obstruction should be scrutinized for signs of a strangulating obstruction when the clinical history and physical findings suggest its presence. From the experiments of Chesterman, it would seem that venous return from a trapped loop of intestine may be impeded without a concomitant block of the bowel.

itself. Among the less frequently seen signs of strangulating obstruction on the survey film are long fluid levels and large amounts of fluid in the loops or solitary segments of bowel distended out of proportion to the remainder of the intestinal tract. These suggest the possibility of strangulation and a search should be initiated for the previously described signs as well as for the clinical confirmation of the diagnosis.

Not all of the various radiologic signs which suggest strangulating obstruction can be expected to appear in any one case. There may be only one of these signs such as the coffee bean sign or the pseudo tumor sign or fixation of the intestinal loops, to lead one to a correct radiologic diagnosis. Furthermore strangulating obstructions may present findings that simulate or are simulated by the following conditions:

- 1 A normal abdomen
- 2 Paralytic ileus
- 3 Non-strangulating obstruction
- 4 Peritonitis
- 5 Inflammatory and strangulating tumors
- 6 Mesenteric arterial and venous thrombosis
- 7 Neurovascular conditions

Paralytic Ileus. Paralytic ileus associated with a pelvic abscess or an abscess anywhere in the peritoneal cavity may demonstrate only a single loop or several loops of bowel in the neighborhood of a large homogeneous radiopaque mass. This is suggestive of an inflammatory process. The peritoneal fat line previously described is usually absent in the region of this inflammatory mass. If the peritonitis is generalized there is apt to be distention of the small bowel as well as the colon and the loops of distended bowel are scattered throughout the abdomen. There is usually a very sharp definition of the intestinal gas patterns in the absence of fluid as a result of the marked impairment of peristaltic activity of the bowel. In those cases in which fluid is present within the bowel or is present as an exudate in the peritoneal cavity there is marked thickening of the bowel wall indicated by the fact that the intestinal gas is separated loop from loop by an increased margin. In addition the loops of gas-filled bowel are relatively fixed in position as



FIG. 240 Marked small bowel distention and gas in the colon. This is evidence of paralytic ileus. Notice increased density in the pelvis. Patient had a pelvic abscess.

shown by the fact that changes in position with a ray at hourly intervals produce little or no change in the gas pattern. It is essential that patients with intestinal obstruction or suspected intestinal obstruction be examined carefully and repeatedly. Radiologic examinations must be continued at frequent intervals until an exact diagnosis can be made.

Occlusive Vascular Disease. Intestinal obstruction as a result of mesenteric vascular occlusive disease presents radiologic findings that are extremely difficult to interpret correctly. In general the radiologic findings are very scanty in contrast to the marked clinical or operative findings. However repeated survey films of the abdomen may furnish valuable information as to whether the obstructive process is improving or becoming worse. From the radiologic point of view if the small bowel becomes more distended and the loops become more definitely visible with the passage of time there is presumptive evidence of an increasing degree of obstruction and surgical



FIG 241 Small bowel distention and exudate present in the abdomen as shown by the fact that there is thickening of the intestinal walls.



FIG 242 A mass in the pelvis with fluid in the abdomen. The patient suffered from volvulus of the sigmoid. Notice the homogeneous appearance on the survey film making diagnosis using air in the gastrointestinal tract a contrast medium a difficult process.

intervention is suggested. If on the other hand the distended loops become smaller and the amount of gas in the colon increases, it would suggest that there is a decrease in the degree of obstruction or that the obstructive process is incomplete. The radiologic signs associated with occlusive vascular disease may demonstrate gas accumulations in the bowel which may be slight for a considerable period of time. The distribution of the gas is often bizarre in appearance. The gaseous distention increases since there is no venous drainage to carry it off. The entire small bowel and the colon up to the splenic flexure are generally involved in the distending process when the occlusive vascular disease involves a major vessel. The radiologic signs at this time may demonstrate gaseous dilated loops of bowel fully as large as those seen in intestinal obstruction. Fluid levels develop within the gas distended bowel but these are scanty and are due to blood. Although at first the pattern of gas distended bowel is very peculiar as the process continues the distention assumes the size and pro-

portions of intestinal obstruction. In general the radiologic findings are not in proportion to the marked clinical findings of a critically ill patient. When the occlusive vascular disease is venous in origin the edematous loops of bowel and the abdominal fluid produce a dense homogeneous shadow throughout the abdomen. Gas distended loops of bowel are usually absent. A few small collections of gas may be present but these are minimal in extent.

Intussusception Although the value of survey film in cases of suspected intussusception is chiefly to establish whether an intestinal obstruction is present nevertheless certain characteristic signs of intussusception may be recognized.

- 1 A soft tissue shadow of the intussusception may be visible.
- 2 Gas in the loop of bowel entering the intussusception may be funnel shaped.
- 3 The apex of the intussusception may be visible.



FIG 243 Loops of bowel separated by exudate. The loops of bowel on repeated films were shown in a relatively fixed position. This is indicative of an exudate in the peritoneal cavity indicating peritonitis.

ble as a soft tissue density producing a concave filling defect in the air column immediately distal to it.

- 4 A crescent shaped gas shadow capping the apex of the increased density shown to be intussusceptum is indicative of intussusception.
- 5 Gas may be seen in the lumen of the bowel outlining the intussusceptum between it and the intussusceptans.

Gallstone Ileus In former years a correct preoperative radiologic diagnosis of intestinal obstruction due to gallstones was made in very few cases. In reviewing the literature on this subject in 1937 Borman and Rigler reported only seven previous case reports of this type. The chief reason for this was the failure to recognize the possibility of biliary calculus producing obstruction. In addition the reluctance of the surgeon to permit the use of barium by mouth in cases in which intestinal

obstruction was a possibility often made it impossible for the radiologist to determine the cause of the obstruction.

The chief radiologic findings indicating gallstone ileus are

- 1 Visualization of gas in the biliary system indicating a fistula between the biliary and gastro intestinal tracts.
- 2 Gallstones that are radiopaque may occasionally be visible in radiographs of the abdomen.
- 3 The radiologic evidence of partial or complete intestinal obstruction may be associated with a clear cut history of biliary tract disease. In such cases a cholecystogram is desirable so that stones in the gall bladder may be demonstrable suggesting the presence of gallstone ileus.

It has been estimated that approximately 80 per cent of the stones which are actually present and cause intestinal obstruction cannot be seen on the survey film of the abdomen regardless of whether the stones are in the bowel or in the gall bladder or common duct. This is due to the presence of cholesterol and the absence of radiopaque minerals. As a result the single most important sign indicating the presence of a cholecystenteric fistula which suggests the possibility of gallstone ileus is the presence of gas in the biliary radicals.

Obstruction of the Colon The presence of a gas pattern suggesting continuity in the normal colon must be differentiated from the continuous pattern formed by the obstructed small bowel. The small intestine may be readily identified by the presence of Kerkring's folds which extend completely across the lumen of the bowel creating a herringbone appearance. In the colon the plicae semilunares extend only partially across the lumen. If intestinal distention has reached a marked degree Kerkring's folds disappear from the radiograph. This makes it very difficult to distinguish between the large and small bowel. However loops of distended bowel at the edges or the sides of the abdomen are most likely to be colon whereas the small bowel loops generally take the center of the abdomen. In case of doubt it may be necessary to resort to a barium enema.

Small bowel distention although generally of lesser degree than colonic distention may so dominate the radiograph when the lesion is in the cecum or ascending colon that in error in diagnosis of small bowel obstruction may result.

Aseroft and Samuel call attention to the fact that the colon may be so filled with fecal material that the characteristic gas shadows are absent. This is particularly apt to occur in the descending colon because the fecal material is in mass and semi-solid by the time it reaches the descending colon. In neglected cases fecal material has been found to involve the entire colon and the cecum presenting a typical pebbly appearance indicating inspissated feces.

Among the most difficult cases to diagnose from the survey film alone are the so-called no gas types of obstruction. In these cases the survey



FIG 245 Note the marked intestinal distention in the presence of obstruction of the colon with an incompetent ileocecal valve. This is a lateral view.



FIG 244 Obstruction of the rectum as a result of a huge fecal impaction. Notice the speckled appearance of the fecal material in the right colon. It was impossible to introduce barium and visualize the colon because of this huge mass of feces.

film shows no gas in the colon and practically none in the small bowel. The absence of gas in the colon in these patients immediately creates a suspicion of mechanical small bowel obstruction.

When an intestinal obstruction is established in the descending colon the time of appearance of intestinal gas shadows in the small bowel is extremely variable depending upon the competence of the ileocecal valve. In general when the ileocecal valve is incompetent intestinal gas is observed 8 to 10 hours after the onset of acute obstruction. In those cases in which the ileocecal valve is competent there may be enormous distention of the right colon and cecum with little or no gas present in the small bowel. In any case of small bowel distention with gas as shown on the survey film the abdomen should be auscultated. With the stethoscope one may determine with some degree of accuracy whether the intestinal



FIG. 246 Same patient as in Figure 245. Obstruction of the rectosigmoid due to carcinoma with an incompetent ileocecal valve. Note marked intestinal distention.

distention is on a mechanical or a paralytic basis.

Pelvic tumors may sometimes prevent gas from entering the rectum even in the absence of a complete intestinal obstruction. Any inflammatory or hemorrhagic process may so compress the rectum at the rectosigmoid as to give a false impression of the flaccidity of the rectum and its ability to retain gas. Occasionally isolated gas distended loops of bowel may occur without any obstruction as a result of a spastic ileus. In such cases the loops of bowel are always smaller than in intestinal obstruction and there is usually no associated pain.

A survey film of the abdomen in the presence of obstruction of the left colon may at times show such tremendous distention of the small bowel that it masks the colonic distention. In cases of this type if an intestinal decompression tube were passed and the small bowel decompressed the colonic distention becomes quite obvious on the survey film. In an occasional instance the small bowel may appear to be tremendously distended

and little or no distention may be found in the colon. This does not obviate a diagnosis of obstruction of the colon if the history is fairly typical of such obstructive colonic lesions. In such cases one must always correlate the survey film with the clinical history and physical findings. In cases in which a history of colonic obstruction is suspected a barium enema should be performed following a sigmoidoscopic examination. By so doing the diagnosis can often be established beyond much doubt.

Cecal Volvulus. The typical radiographic appearance of cecal volvulus demonstrates a distended cecum of variable size with one or two fluid levels when the film is taken with the patient in the erect position or in the lateral decubitus. The unusual dilatation of a segment of bowel containing gas and fluid is the one feature which is immediately noted on examination of the survey film. In those instances in which there is displacement of the cecum from the right lower quadrant the absence of a cecal gas shadow from this location and the presence of a dilated bowel in an abnormal situation should lead one to suspect the presence of cecal volvulus.

The radiologic findings noted on the survey film are variable depending upon whether the obstruction is complete or incomplete. In those instances in which the obstruction is complete the distention of the cecum may be quite marked although the involved cecum and ascending colon may be present in the right flank and in the right lower abdomen in a normal position. There may be an absence or marked diminution of gas or fecal material in the colon distal to the point of obstruction. Ileus or intestinal distention of the small bowel of variable degree may be found. Occasionally there is a reversal of the lateral convex border of the cecum and the ascending colon which may suggest abnormal mobility and torsion of the right colon. Serial roentgenograms are necessary to demonstrate abnormal mobility or migration of the right colon in any case in which volvulus of the cecum is suspected. In those cases in which the cecal volvulus occurs on its longitudinal axis there may be all the signs of complete obstruction without displacement of the segment. In such cases showing complete obstruction it is apparent that the cecum must have twisted at least 360 degrees

on its long axis. In most cases of cecal volvulus there is gross displacement of the cecum and ascending colon. It is generally thought that 40 to 60 per cent of all cases of cecal volvulus present the distended cecum and right colon in the left upper quadrant. In this location they may be confused at times with a dilated stomach. The completeness or incompleteness of the intestinal obstruction associated with cecal volvulus depends more upon the tightness of the twist than upon the number of turns which the twist undergoes.

The length of the dilated cecal segment and the right colon determines their appearance on the X-ray and also determines their ectopic location. In those cases in which the obstruction is complete the markings are lost because of the tremendous pressure developed in the obstructed short cecal segment which functions as a closed loop in the presence of a competent ileocecal valve. The probability of ectopic location of the twisted cecum increases with an increase in length of the right colon undergoing volvulus.

In an occasional case with an incompetent ileocecal valve the terminal ileum may be found lying to the right of the distended cecum. In an occasional instance a gradual narrowing of the gas distended bowel to a cone may be demonstrated by the intestinal gas patterns. This suggests twisting of the mucosa. In those cases of volvulus of the right colon in which the obstruction is incomplete the clinical as well as radiologic findings are those of an incomplete intestinal obstruction. In such cases the colon distal to the point of obstruction may contain appreciable amounts of gas or fecal material. As a result it may be difficult or impossible to localize the exact site of obstruction on the survey film alone. The use of a barium enema is essential for a correct diagnosis when this occurs.

The diagnostic criteria set down by Fiegel and Finkel for partial torsion with incomplete obstruction are as follows:

1. Cecal torsion should always be considered when the degree of cecal distention is out of proportion to the dilatation of the remainder of the colon.
2. Considerable fluid is found within the cecum.
3. The cecum and ascending colon normally



FIG. 247 The use of the long intestinal decompression tube to decompress the small bowel in the management of obstructions of the colon due to carcinoma. In this patient we see an example of a high grade partial obstruction of the terminal ileum approximately 10 cm from the ileocecal valve. This was not shown radiographically despite the reflux of barium. In fact the radiologist noted that the barium showed no evidence of obstruction in the area 10 cm proximal to the ileocecal valve. This indicates the fallacy in depending upon radiologic diagnosis by means of barium enema for obstructions in the terminal ileum beyond the point 10 cm proximal to the ileocecal valve.

show a convex lateral border and a concave medial border assuming a somewhat kidney shaped appearance. In an occasional case the twisted right colon maintains its kidney appearance in spite of severe distention. In this event partial torsion will demonstrate that the lateral border is concave. Cases of partial torsion without displacement may present an apparent discontinuity of the gas column slightly distal to the dilated cecum. This may suggest the point of torsion.

4. Some degree of gaseous small bowel distention may be found.

Interposition of Colon (Chiladiti's Disease)

Hepatodiaphragmatic interposition of the colon is a condition in which a redundant colon comes to lie between the superior surface of the liver and the right dome of the diaphragm. The presence of a gas filled viscus in this location may be misdiagnosed as subphrenic accumulation of air indicating a perforated viscus or it may suggest the possibility of volvulus. Balloon like distention of both the hepatic and the splenic flexures commonly occurs and pronounced redundancy of the sigmoid colon may be associated with such ballooned out flexures. The condition (Chiladiti's disease) is an innocuous one and was first described in 1910 by Chiladiti for whom it is named. The condition requires no treatment and its only significance is that one should be able to recognize its presence and to differentiate it from the more serious lesions capable of producing gas filled shadows below the diaphragm.

Small Bowel Obstruction in Infancy and Childhood The radiologic diagnosis of small bowel obstruction in infants is very difficult. The basis for a diagnosis of intestinal obstruction in older children and adults is the finding of intestinal distention with gas or gas filled loops in the small bowel where normally no gas is found. In infants however such a condition is not abnormal. The finding of intestinal gas in the small bowel up to the age of two years is normal. Gas appears in the stomach almost immediately after birth even when the infant is not fed during the first 12 hours of life. The studies of Wasch and Marek demonstrate that the proximal portion of the small intestine contains gas within the first hour of life. At the end of three hours of life the entire small bowel contains gas. Segments of large intestine may also be visualized as a result of their gaseous content. Eight hours after birth there is a relatively large amount of gas in the small intestine as compared with the colon. At the end of 12 hours the balance shifts so that the colon is the most prominent.

A survey film of the abdomen in infancy discloses that the normal small bowel containing gas is a formless and shapeless mass with a hazy washed out appearance. Intestinal obstruction in infancy however causes this shapeless formless mass of gas filled loops to assume a bulge and as



Fig. 248 Gas filled small bowel loops in a newborn

a result of pressure from the adjacent bowel to take a square shape. This is called "squaring off." At first glance it would appear that the bowel had become distended with gas. The pressure of one

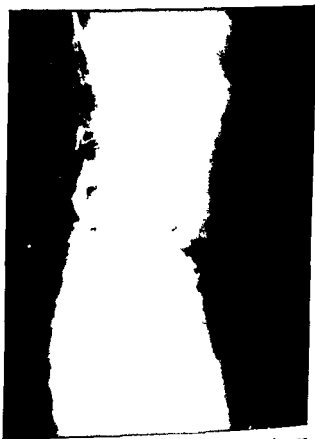


Fig. 249 Lateral view of an 11-year-old child showing small bowel distention indicative of intestinal obstruction

distended loop against another creates the small bowel to form a square. As the intestinal gas increases the radiologic appearance of the bowel becomes blacker like that found in any organ in which the amount of gas increases. With an increase in the intraluminal gas the bowel becomes distended and the continuity of the segment of small bowel becomes established radiologically. When these continuous loops of bowel turn upon themselves the so-called hairpin turn are noted. As the distention increases the loops of bowel may be noted to lie one above the other a phenomenon called *lavering*. This *lavering* may appear in cross sections as a tier of squared off loops.

Lavering is a later manifestation of small bowel obstruction which ultimately may be associated with the formation of fluid levels in the bowel. In any case in which there is doubt as to the pre-

ence or absence of intestinal distention repeated survey films of the abdomen should be taken at hourly or bi hourly intervals at which time the progressive increase in intestinal distention may be noted.

The radiologic diagnosis of congenital duodenal obstruction is usually made with barium as a contrast medium or by means of a survey film of the abdomen in which swallowed air is used as a contrast medium. Although many doctors believe that one should not hesitate to use thin mixtures of barium in upper gastro intestinal series to demonstrate congenital duodenal obstructions nevertheless there are many others who warn of the dangers inherent in the use of such contrast media in the newborn. Among the dangers is the possibility that the barium may plug an anastomotic stoma which may be created in a short circuiting procedure. In addition because of vomiting some of the barium may be aspirated and thus produce a very serious type of aspiration pneumonia. Swenson and Ladd believe that barium should be reserved for rare conditions dealing with infants and should not be used for intestinal obstruction and its diagnosis.

In those cases in which barium is not used as a contrast medium a survey film of the abdomen may demonstrate intestinal obstruction by virtue of the gas or air shadow which is present. In these cases air is seen to fill that portion of the gastro intestinal tract above the point of obstruction while little or no gas may be visible in the intestine beyond. A limitation to this method of diagnosis of obstruction is the finding that during the first 12 to 24 hours after birth the stomach and duodenum may only be slightly dilated. In such cases the duodenal curve may be poorly outlined by air.

Mellins and Milman proposed a simple method whereby a diagnosis can be made in such cases using air as a contrast medium. Their diagnostic procedure is based upon the observation of Soveri who noted that if 200 to 300 cc. of air were introduced into the stomach of infants the empty stomach could evacuate this quantity of air into the intestines within a half an hour. Much of the air passed through the stomach within five minutes and if the intestine was empty the air passed completely through it in one to two hours. Because of



FIG 250 Same patient as in Figure 249. Distended small bowel loops noted on the survey film. At surgery the patient was found to have volvulus of the small bowel as the result of an appendectomy three days previously. Detorsion of the volvulus resulted in uneventful recovery.



FIG 251 Marked small bowel distention with little or no gas in the colon. This is suggestive of a small bowel mechanical obstruction.



FIG 252 Note the marked small bowel distention. A Cantor tube was successfully passed to decompress the small bowel. A diagnosis of paralytic ileus was made on clinical grounds. The fallacy of depending upon radiologic findings exclusively is thus apparent in this case.

20 Upon examination he was seen to be an elderly man and he appeared to be acutely ill. The only relevant physical findings were confined to the abdomen. There was diffuse tenderness over the entire abdomen and marked distention. No peristaltic activity was audible by auscultation. No fluid wave or shifting dullness could be demonstrated. On rectal examination a doughy very tender ill defined mass was palpable in the pelvis.

A preliminary survey film of the abdomen taken May 8 showed marked distention of the small bowel. A small amount of gas was noted in the cecum and none in the remainder of the colon. On this basis a roentgen diagnosis of mechanical small bowel obstruction was made (Figure 251).

On the basis of the physical findings a diagnosis of paralytic ileus due to a pelvic abscess was made despite the roentgen diagnosis. A Cantor tube was passed and intestinal intubation begun. Intravenous fluids were given to restore the altered fluid and electrolyte balance and penicillin and streptomycin were given.

On May 9 a preliminary film was again taken with the report as follows: An intestinal tube has been inserted. Very little change has taken place as yet in the number of distended loops of small bowel. Conclusion: Cantor tube in jejunum. Mechanical small bowel obstruction (Figure 252).

As a result of treatment with intubation antibiotics

and the restoration of fluid and electrolyte balance the patient improved daily. On the eighteenth hospital day he developed a fluctuant mass in the pelvis which ruptured into the rectum discharging a large amount of foul purulent material. His recovery from that day was rapid. He was discharged as cured on June 3 1949 his twenty-seventh hospital day. The final diagnosis: Pelvic abscess with paralytic ileus.

This patient presented all the characteristic radiologic finding for a diagnosis of small intestinal obstruction. A roentgen diagnosis of mechanical small intestinal obstruction was made and operation advised. On clinical grounds a diagnosis of paralytic ileus due to pelvic abscess appeared most likely. Despite the absence of gas in the colon it was believed that an unusual gas pattern was present in this case and that it constituted an exception to our usual roentgen diagnostic criteria. The final diagnosis of paralytic ileus was proved by the clinical course of the disease. Surgical intervention in this case might have been most serious.

Case 2

A.A. a female aged 50 was admitted to the hospital on July 20 1949 complaining of vomiting and cramping pain

in the abdomen. The pains began insidiously in the mid portion of the abdomen about 10 days prior to admission. Vomiting followed shortly thereafter. She had vomited almost all of her food for the past week. She noticed that she had been more constipated in the past month and had no bowel movement for several days prior to admission. She had never noticed bloody or tarry stools although she had recently had some trouble with hemorrhoids which she attributed to straining. Physical examination showed no significant findings except for the abdomen which was distended and tympanic but not tender. No fluid wave or masses were detected. Peristaltic sounds were diminished and heard only in the right lower quadrant. Pelvic examination showed no pathology. Rectal examination showed grade 2 internal and external hemorrhoids. Digital examination showed the rectum to be filled with soft feces. After adequate preparation a sigmoidoscope was passed 25 cm. No mucosal lesions were found. A preliminary film of the abdomen on July 26, 1949, showed considerable gaseous distention of the small intestine with no gas visible in the colon. The roentgen diagnosis was

marked low obstruction of the small bowel probably ileum (Figure 253).

On July 27 the abdomen was opened. The entire small bowel was found to be dilated down to the ileocecal junction. The cecum contained only a small amount of gas while the ascending and transverse colon which were immediately visualized were contracted and empty. While the cecum was being palpated the gas from the ileum passed into it and caused it to dilate up to the hepatic flexure. This area was then mobilized in search of a tumor and during this manipulation, the transverse colon filled with gas. Palpation was then continued down the descending colon which was still contracted and contained soft stool which increased in amount down to the mid sigmoid. There a napkin ring type of tumor was found to cause a high grade obstruction. The bowel below the tumor was collapsed and emptied. By the time this exploration was completed the entire colon down to the tumor in the mid sigmoid was distended with gas. A transverse loop colostomy was done and the abdomen closed. There was no drainage. The final diagnosis was carcinoma of the sigmoid colon with obstruction.

On August 22 a resection of the sigmoid colon and an end to end anastomosis were performed. This was followed by a resection of the stoma of the colostomy and anastomosis on September 2, 1949. The patient made an uneventful recovery.

This patient proved to have an annular obstructing carcinoma of the pelvic colon. Roentgenologically she should have shown considerable distention of the colon with or without gas in the small intestine. Actually however she presented the roentgenologic findings of a marked small intestinal distention with no gas in the colon so that a roentgen diagnosis of small bowel obstruction was made. At operation the colon was found to be collapsed and free of gas while the small bowel was dilated. It is difficult to explain this condition on a mechanical basis. The intestine was apparently in a sympatheticotonic state with peristalsis of the ileocecal valve and a hypotonic and dilated small intestine. As soon as the ileocecal valve was palpated in searching for the cause of the obstruction the gas began to pass into the cecum and through the colon to the point of sigmoid obstruction.

These cases indicate that the use of intestinal gas patterns as a diagnostic method is not free from error. At times bizarre gaseous intestinal patterns appear that tax to the utmost the diagnostic acumen of the radiologist. In such cases further study by barium enema or small intestinal



FIG. 253 Note the marked degree of small bowel distention with little or no gas in the colon suggestive of mechanical small bowel obstruction. This patient was found to have an obstruction of the colon.

study by means of barium suspension injected through an indwelling long intestinal tube would seem to be indicated before a definite diagnosis is made.

RADIOLOGIC DIAGNOSIS BY OTHER MEDIA

When a diagnosis of intestinal obstruction has been made on the survey film, the radiologic study should properly begin with a barium enema if it is possible. At times this may not be possible because the condition of the patient is so precarious that a barium enema cannot be given. It is always desirable to have a sigmoidoscopic examination precede the barium enema since approximately 65 per cent of all colonic obstructions occur within 25 cm. of the anus. The majority of these obstructions can be diagnosed sigmoidoscopically without requiring the use of barium enema study. If a barium enema is performed, the barium should be introduced gently and without undue force because in many cases a partial obstruction has been made

complete by forcing the contrast medium beyond the obstructive lesion where it becomes inspissated thus producing a complete obstruction proximal to the point of narrowing. The presence of an accumulation of inspissated barium above an obstructive colonic lesion has been the cause of considerable inconvenience to the surgeon and has at times resulted in serious consequences to the patient.

Barium

Barium sulfate is widely used as a contrast medium in the diagnostic study of the gastrointestinal tract either as an enema to visualize the colon or orally to visualize the upper gastrointestinal tract. In addition to its use as a contrast medium to visualize and diagnose obstructions of the colon it has also been found useful in the treatment of ileocolic intussusception under fluoroscopic control.

Barium Enema Used Diagnostically In the case of obstructing lesions of the sigmoid colon



FIG 254 Marked small bowel distention associated with obstruction of the rectosigmoid due to carcinoma.



FIG 255 Diverticulosis. Diverticula are well shown by barium enema study.

it may be difficult to distinguish between carcinoma of the colon and obstructing diverticulitis. However, if some of the barium were to pass proximally or if other diverticula are visualized with the barium enema, one might assume that the obstructing process is diverticulitis. However, it must be borne in mind that in an occasional case diverticulitis or diverticulosis of the colon may be associated with a carcinoma producing the obstruction. In such cases the diverticulosis would be only an incidental finding and should not lead one astray.

In a similar fashion obstructive lesions of the ileocecal region may prove to be tuberculomas. This would be suggested by evidence of tuberculous nodes noted on the preliminary chest X ray.

Redick and Harrington have suggested the use of a barium enema in the diagnosis of mesenteric vascular occlusion. This technique is based upon the frequent finding of mechanical obstruction at splenic flexure radiologically demonstrated whenever there is occlusion of the superior mesenteric artery. In such cases Redick and Harrington suggest that a barium enema would be of value since a presumptive diagnosis of mesenteric vascular occlusion may be made if the column of barium passes through the area radiologically simulating obstruction noted on the survey film. The experience with this technique is very limited at the present time although it is possible that it may be the best single test for differentiating a true intestinal block from the stasis resulting from vascular occlusion. It is unfortunate that in those cases in which the main mesenteric vessels are obstructed the patients are usually in such a degree of shock that their condition precludes radiologic studies by means of barium enema.

In general it may be said that the use of a barium enema gives clear cut evidence for obstruction of the colon. In addition the method permits a diagnosis of the degree of obstruction whether it is complete or incomplete. The exact location of the obstructive process is also shown. Thus, when properly used, barium enema is a safe and effective method of diagnosing obstructive lesions of the colon, permitting accurate diagnosis of the type of obstruction as well as its degree and location.

Barium Enema Used Therapeutically In

intussusception the barium enema is not only a diagnostic measure but may be a therapeutic one as well. A survey film of the abdomen may disclose intestinal obstruction but it may be difficult or impossible to be certain of the presence of intussusception from the survey film alone. In intussusception involving the colon or ileocecal region or in the colocolic type of intussusception there are many who advise reduction of the intussusception by means of barium enema under fluoroscopic control. This method was popularized by Hirschsprung who used it with excellent results. There is little doubt that the use of barium does have its place in the reduction of the ileocolic or colocolic type of intussusception. The ileocolic invagination is not easily reduced by this method and of course in small bowel intussusception the method is of no value at all. Since approximately 70 per cent of the intussusceptions in children are of the ileocolic variety the barium enema method may be effective if the child is seen relatively early. In late cases because of the adherence between the intussusceptum and the intussusceptans surgical intervention is essential.

Barium Sulfate Given by Mouth for Diagnosis of Obstruction There is considerable variety of opinion as to the advisability of giving barium by mouth even in small amounts to aid in the diagnosis of intestinal obstruction. There are those who are vehemently opposed to the use of any form of barium taken by mouth in the diagnosis of obstructive lesions of the gastrointestinal tract. On the other hand there are equal numbers of competent radiologists who are ardent proponents of the use of small amounts of barium by mouth for diagnostic purposes. Between the two extremes there are many who use very small amounts of dilute barium in selected cases to aid in the diagnosis of intestinal obstruction.

Opponents to use of oral barium Welter is quite vehement in his objections to the use of orally administered suspensions of barium for the diagnosis of intestinal obstruction. He believes that oral suspensions are contraindicated when the clinical and radiologic evidence suggest that the obstruction is complete or nearly so or even when it is low or moderate in degree or is situated in or below the lower two thirds of the small bowel.



FIG 256 Small amount of dilute barium given by mouth visualize the gastro intestinal tract. Note the marked small bowel distention.

Some of Weber's objections to the use of barium by mouth are

- 1 Any opaque fluid added to the fluid and gaseous content of distended intestinal loops is certain to be widely dispersed unevenly mixed and diluted to the point of losing adequate contrast
- 2 The greater the degree of distention the more effectively intestinal mobility is reduced and as a result the time required for radiologic examination is likely to be extended beyond practical limits
- 3 The use of the intestinal decompression tube as a radiologic diagnostic instrument is of little or no value since too much time often several days may be required before the intestinal decompression tube can be passed to the point of obstruction

Proponents of Use of Oral Barium In opposition to the position taken by Weber Case believes

that the use of small amounts of barium in cases of suspected acute small bowel obstruction is harmless. The reason for using small amounts of barium in such upper gastro intestinal studies is that the obstructed bowel with the barium in it can be detected very easily by visual examination. In addition the presence of the barium filled loop can be very easily detected on the X ray so that the exact point of obstruction may be noted. Case calls attention to the fact that great care must be exercised in giving barium by mouth in any case in which an entero anastomosis has been performed.

There are many who pursue a middle of the road course with regard to the use of barium suspensions by mouth in cases of intestinal obstruction. Frimann Dahl feels that the use of barium in cases of intestinal obstruction depends upon the individual problem presented and that it should not be used routinely unless the close cooperation of the surgeon is available. In high intestinal obstruction since very little gas may be found in the small bowel proximal to the point of obstruction Frimann Dahl is of the opinion that barium may be given fairly early in order to make a diagnosis of obstruction. Radiologists of wide experience are agreed that barium may be used when the survey film is not adequate and that if given in small amounts it will not inspissate and will not increase the degree of obstruction. As a matter of fact quite the opposite occurs because in intestinal obstruction there is usually so much fluid proximal to the point of obstruction that as the dilute barium descends it is further diluted and gradually becomes thinner and thinner ultimately becoming quite transparent. By using small amounts of barium in this fashion such definite radiologic evidence may be obtained that even though some time is lost in this radiologic study nevertheless the ultimate results are worthwhile since many cases are spared surgical intervention. In those cases in which the obstruction is incomplete a small amount of barium may be massaged beyond the partially obstructing process so that its nature may be determined with a relative degree of exactitude. In the presence of a complete obstruction however the barium will come to rest at the point of obstruction. It must

be remembered also that at times local spasm associated with neighboring disease may result in such an obstruction to the flow of barium that it simulates complete intestinal obstruction.

The range of opinion in regard to the use of barium by mouth in the diagnosis of obstructions appears to be chiefly the result of a failure to realize that only very small quantities of barium need be used for such studies. The dangers of giving the barium meal to patients with suspected intestinal obstruction have been stressed repeatedly, few radiologists would knowingly give barium in large amounts or high concentrations to any patient suspected of having intestinal obstruction.

In those cases of intestinal obstruction in which it is desirable to give barium by mouth usually not more than 4 ounces of well diluted barium are administered. Administration of such small amounts of barium by mouth is indicated for those individuals in whom the clinical evidence suggests mechanical obstruction but the radiologic survey film shows little or nothing. By giving such small amounts of barium it is easy to see the fluid filled loops of small bowel which generally sink down in the lower abdomen and appear as widely dilated intestinal loops. If the obstruction is incomplete some of the barium may go through the point of obstruction and appear in the cecum, yet some dilatation of the small bowel is often found proximal to this obstructing point.

Dangers in Administration of Barium. One of the chief dangers in the administration of contrast media for the diagnosis of obstruction is the length of time which may be required to carry on an adequate radiologic study. The patient may have a compromise of the circulation and strangulation of the bowel long before the intestinal studies are completed.

The use of large amounts of barium in the concentration normally used in gastro intestinal studies must be avoided in any patient in whom bowel obstruction is suspected. Even though the fluid filled obstructed bowel loops do dilute the ingested barium to some extent the settling out of a thick barium mixture may convert a partial bowel obstruction into a complete one. This is especially apt to occur in any patient in whom a bowel resection has been performed. In cases of this type the

anastomosis may become obstructed by a thick barium mixture. The barium may become putty like as a result of a settling out of the barium from its suspension due to standing within the gastro intestinal tract or it may become putty like as a result of absorption of water in those cases in which little fluid is found in the gastro intestinal tract. In either case such a putty like mass of barium could effectively plug up a partially obstructed bowel. Case reported one patient in whom a fatality resulted because a mass of barium obstructed the lumen at the site of anastomosis.

Despite the universal acceptance of the dictum that barium sulfate suspension in its normal concentration and amount should not be given for the performance of an upper gastro intestinal series when obstruction of the bowel is suspected such barium suspensions may be administered because of a failure to diagnose or even suspect the presence of a partial bowel obstruction. The complete obstruction which may result would then be very difficult to correct. Not infrequently the barium remaining after routine upper gastro intestinal series may produce bowel obstruction some days after the radiologic study. Such cases have been reported by Klein who reported a fatal ileus from contrast medium suspension by Golob who reported the advisability of immediate colonic irrigation following barium enema by Orgel who reported barium meal simulating intestinal obstruction and by Straker who reported barium as a factor in intestinal obstruction.

The Cantor tube with an 18 Fr. lumen provides an ideal instrument to remove barium sulfate suspensions given by error in the type of case mentioned. In one instance an upper gastro intestinal series was ordered because of the failure to recognize the presence of a high grade partial bowel obstruction. The radiologist noted a greatly distended barium visualized small bowel due to a complete bowel obstruction (see Figure 27). This was the result of the barium sulfate suspension converting an incomplete obstruction into a complete one. This same effect has been reported by the too forcible injection of barium sulfate as an enema in the diagnosis of obstructing lesions of the rectosigmoid. Many such cases have been reported. One of the recent ones reported by

Byronofsky required primary resection and end to end anastomosis for the acute large bowel obstruction which resulted. In such cases the barium sulfate may be forced through an incomplete obstruction of the rectosigmoid or sigmoid to form putty like masses proximal to the stenosing lesion. A complete obstruction would then result.

The treatment of such barium induced bowel obstructions generally consists of giving large amounts of mineral oil by mouth in an effort to soften the barium mass kneading of the mass of barium through the abdominal wall high colonic irrigations and proctoscopic dislodgement with removal of such masses in the pelvic colon. Fatal cases of ileus as a result of barium impaction have been reported.

Using a single lumen simplified intestinal decompression tube (Cantor) to remove barium sulfate suspensions from the small bowel was found to be a simple safe and effective procedure. In 48 hours the barium suspension producing small bowel obstruction may be easily diluted and evacuated through the Cantor tube. The following case is an excellent example of the efficiency of this method of treatment.

Case report

K.H. a 64 year-old woman was admitted to the hospital January 9 1949 with a diagnosis of gallbladder disease. Her chief complaint was right upper quadrant and epigastric pain. She had been comparatively well until September 1948 at which time she had an attack of epigastric pain which was knife like in character and more severe in the right upper quadrant. About two weeks before she began to vomit periodically. This was often associated with diarrhea. The latter would invariably relieve her right upper quadrant pain. Olive oil and orange juice also relieved her pain somewhat. At times the pain was generalized over the entire abdomen. The vomiting and diarrhea subsided shortly after Christmas of 1948 but the epigastric pain continued. She now noted difficulty in keeping solid food down. The pain increased in severity. For this reason hospitalization was advised.

On examination the patient was found to be an elderly white woman in her sixties. Temperature 99 degrees pulse 100 respirations 24. The only positive physical findings were related to her abdomen which was found to be soft and not distended. There were no masses. Tenderness and rigidity with some spasticity were noted in the right upper quadrant and in the epigastric region. Metallic bowel sounds were audible in the upper abdomen. The liver spleen and kidneys were not palpable. Her urine was negative. Her RBC 4 450 000 hemoglobin 84 per



FIG. 257 Note the marked distention of the small bowel as a result of the erroneous ordering of an upper gastro intestinal series in the presence of a high grade partial intestinal obstruction.

cent (14 grams). Her WBC 5400 with polymorphonuclear leukocytes 71 per cent filamented 60 per cent nonfilamented, 6 per cent and lymphocytes 29 per cent. Her Kahn was negative. An admission diagnosis of chronic cholecystitis was made.

On January 11 1949 the patient was given barium sulfate suspension for a routine upper gastro-intestinal series. X rays taken showed the contrast medium gradually progressing in the small bowel and diluting markedly with large quantities of retained fluid material in the jejunum and ileum so that ultimately the dilution became so great in the pelvic area and right iliac region that it was impossible to distinguish individual loops and the mucosal structure of these intestinal segments. Obviously this patient presented a low obstruction of the small bowel.

During the days from January 11 1949 to January 14 1949 a Cantor tube was passed successfully and the bulk of the contrast material removed from the intestinal tract each day with a considerable amount of liquid and particulate material. To remove the barium it was necessary to irrigate the bowel with 3000 cc of water daily in order to keep the barium suspension sufficiently diluted so that it could be aspirated by the tube.

On January 14 1949 radiographic study revealed only



FIG 258 The same patient as in Figure 257 Notice the tremendous intestinal distention with obstruction produced by the barium meal A Cantor tube was successfully passed and suction applied Note the presence of the tube far down the gastro intestinal tract



FIG 259 The same patient as Figures 257 and 258 Note the marked decrease in intestinal distention and the almost complete removal of barium from the gastro-intestinal tract

minimal amounts of contrast medium remaining within the bowel The tip of the Cantor tube was in the terminal ileum

On January 15 1949 under spinal anesthesia a right rectus incision was made Upon opening the abdomen the terminal ileum and about 4 inches of the cecum and ascending colon showed evidence of acute inflammation. On examination a congenital adhesive band 4 inches from the ileocecal valve was found to bind the terminal ileum to the posterior abdominal wall This band was cut It was then noted that the lumen of the ileum was constricted by adhesions reaching from the mesenteric border around the entire bowel These were freed restoring the bowel to its normal size The Cantor tube was left in the bowel just above the point of obstruction and the abdomen closed in layers

Following this surgery the patient made an uneventful recovery and was discharged January 25 1949

The management of this type of accident is quite simple and usually effective A long intestinal decompression tube of adequate caliber should be passed down the gastro intestinal tract In this sort of case because peristaltic activity is generally

vigorous in an effort to by pass the obstructing process the tube can usually be passed rapidly The luminal diameter of the intestinal tube should be as large as possible preferably 18 Fr With this type of tube far down the bowel irrigation the bowel through the tube with warm water will effectively dilute the barium suspension permitting its rapid aspiration through the long tube By this simple method a serious situation may readily be converted into a relatively safe one After all the barium has been removed from the bowel and decompression obtained surgical intervention is indicated to correct the cause of the partial bowel obstruction In the case reported a congenital adhesion was found to be the causative element

Umbrathor

Case is enthusiastic about the use of Umbrathor an aqueous preparation of thorium dioxide in all cases of suspected acute or chronic intestinal obstruction He believes that although the adminis-

tration of Umbrathor is harmless should the procedure prove unnecessary it may be extremely time and life saving if obstruction is present. Case reports having used this preparation since 1930 as a substitute for barium in the radiologic examination of suspected obstruction. He uses Umbrathor both orally and rectally.

Umbrathor is a mildly astringent slightly turbid liquid. It is less fluid than water and can be swallowed by the patient or passed through a long intestinal decompression tube. It gives a shadow of very great density equal to or even better than that obtained with suspensions of barium. It has the additional advantage of remaining liquid so that there is no danger of the opaque medium sedimenting out of solution. Case believes therefore that it presents great advantages over barium. Case has found Umbrathor particularly useful in cases of obstruction of the colon when depiction of the mucosal folds is desired. He gives a 70 cc dose of Umbrathor to which is added an equal quantity of plain water preferably cold water. It may be given in a single dose or in divided doses over a period of an hour or an hour and a half. In those patients in whom the long intestinal decompression tube is used 100 cc of diluted opaque material are injected through the tube and the tube is then clamped for an hour or two. The patient is kept for an hour or two in a position which will facilitate the emptying of the stomach; this requires that the patient lie on his right side almost face down with the foot of the bed slightly elevated.

Use of Long Intestinal Decompression Tube and Dilute Barium in the Diagnosis of Small Bowel Obstruction

Highly accurate radiologic diagnosis of partial as well as complete intestinal obstruction is possible with the combined use of the intestinal decompression tube and dilute barium injected through it. This diagnostic technic is particularly useful in those cases of partial high grade intestinal obstruction in which a correct radiologic diagnosis may not be possible by any other method. Accurate radiologic control is particularly important when the long intestinal decompression tube is used following bowel resection. By means of this technic

the patency of the anastomosis can readily be shown.

Dilute barium and the intestinal decompression tube are also useful in cases of intestinal distention associated with obstructions due to inflammatory processes. In such cases it is often difficult to determine whether the distention is due to paralytic ileus from the inflammatory process or to a mechanical obstruction. Whether the obstruction is mechanical or due to paralytic ileus can be demonstrated with a high degree of accuracy by utilizing exact radiologically controlled methods. This determines whether the treatment should continue along medical lines or whether surgical intervention is indicated.

An analysis of the many cases of intestinal distention requiring intestinal intubation has resulted in an attempt to control all phases of this process with more exactness. Radiologic methods to determine the safe period for removal of the intestinal tube have not been utilized as often as they should nor have they received the attention they deserve. In no other way can the surgeon be informed with such accuracy as to whether it is safe to remove an intestinal tube. There are a wide variety of gastro-intestinal conditions in which such radiologic control is desirable. In many cases it is only in this way that the surgeon can be certain that a partial obstruction is present requiring operative intervention or that no mechanical obstruction exists. In the first instance surgery is imperative; in the second it would be pointless to subject the patient to operative hazards.

The radiologic technic of determining just when an intestinal decompression tube should be removed is quite simple but must be individualized to meet the needs of each case. Generally a suspension of barium 30 to 100 cc is injected through the tube under fluoroscopic control and the progress of the barium down the gastro-intestinal tract is followed fluoroscopically as well as by the use of spot films and survey films. Such observations are carried on at intervals during the first 24 hours. During the first five hours the intestinal decompression tube must be clamped off to prevent withdrawal of the barium suspension. The barium mixture should be quite liquid so that its introduction and removal through the tube are

easily accomplished but it must not be too dilute for adequate visualization radiologically. If the obstruction is completely distal to the tube a definite puddling of barium will be observed at the point of obstruction. If the bowel is only partially obstructed some puddling will occur at the site of obstruction but some of the barium will pass through. Generally such cases can be recognized on the 24 hour film by the presence of puddling at the point of obstruction associated with a thin film of barium in the colon distal to the obstruction. In those cases in which there is no mechanical obstruction the intral fluoroscopic observation will usually show well marked barium deposits in the colon and a 5 hour film will furnish conclusive evidence that no obstructing lesion is present. In an occasional case, as a result of stomal edema at the site of a small intestinal anastomosis the 5 hour film may reveal a thin trickle of barium in the colon but the 24 hour film will show a marked increase in the barium beyond the cecum.



FIG 260 X ray taken five hours after injection of barium suspension shows a large amount of barium in the cecum and ascending colon indicating the absence of small bowel obstruction

Not only is the presence of barium in the colon demonstrable but the caliber of the small bowel can readily be determined by this method. Kinks and stenotic areas without obstruction become quite apparent to the trained radiologist. Spot films are particularly valuable in such cases.

In the event that complete obstruction is found distal to the tube suction is reinstituted to remove the barium suspension. In some cases the barium may be flushed out by irrigation with normal saline after which suction is applied. The patient is then treated surgically. When partial obstruction is observed by the radiologist removal of the barium suspension is not required but the surgeon is advised that operation is indicated at the earliest possible time. In such cases the surgical treatment is generally simple so that when the constriction is released the barium readily passes through.

The following four cases typify the exactitude with which the radiologist can distinguish the obstructed from the non obstructed gastro intestinal tract and the importance of such a distinction.

Cas 1

J.S. a 30 year old white woman was admitted to the hospital complaining of pain in the abdomen. During a curettage on the day of admission the surgeon had perforated the uterus and pulled down a loop of bowel with his curette. Immediately after entering the hospital the patient was prepared for surgery. At operation the blood supply to the loop which had been pulled down was found to be so poor that it was necessary to resect 3 feet of small bowel. An end to end anastomosis was performed. Following the operation the patient was given large doses of penicillin and streptomycin by injection. The next day she began to vomit and her abdomen became markedly distended. There was no abdominal rigidity but some tenderness. Her temperature at this time was 101 degrees pulse 120 respirations 25.

A Cantor tube was passed without difficulty and continuous suction was instituted. A 24 hour X ray showed the tube to be well down the jejunum and the patient had improved markedly. The abdomen remained soft and flat and on the sixth postoperative day 50 cc of barium suspension were injected through the long tube to check the patency of the bowel anastomosis prior to removal of the tube. In the interval study some delay was observed in the passage of the barium into the cecum at the end of five hours. During this period the suction had been stopped and the tube clamped to prevent aspiration of the barium. After the fifth hour suction was resumed. On a 24 hour film taken with the tube still in place a considerable amount of the barium was seen to have passed



FIG 261 Radiologically controlled intestinal intubation Dilute barium is injected through the Cantor tube and the dilute barium followed by X ray



FIG 262 The same patient as in Figure 261 The barium has passed into the right colon and the transverse colon This patient proved to have a paralytic ileus and there was no small bowel obstruction.

through the small bowel and was readily demonstrable in the colon The Cantor tube was then removed

In a case of this type in which a bowel resection is done we have an exact method of checking the patency of the anastomotic site as well as noting the presence or absence of postoperative kinks or stenosis Following a 24 hour film we could assure the surgeon that the removal of the long tube was safe In most cases of this type the 24 hour film demonstrates the greater part of the barium in the colon In that event the tube may be removed at once without the necessity of a 24 hour film The 24 hour film is of the greatest value in those cases of delayed evacuation of the barium

Case 7

H M a 58 year old white man, was admitted to the hospital with a history of rectal bleeding His bowel had moved normally and he had been passing gas freely although he had noticed some gurgling in the abdomen for the past four months In the month preceding admission

he had experienced intermittent pain in the left lower quadrant which was invariably relieved by a bowel movement Except for slight tenderness over the left lower quadrant the physical findings were normal Roentgen studies showed a definite filling defect in the mid sigmoid A diagnosis of carcinoma of the sigmoid colon was made

A resection of sigmoid colon was performed with an end to-end anastomosis Following this operation, the patient made a satisfactory recovery until the fourteenth postoperative day At this time he complained of cramping abdominal pain and the abdomen became distended Upon examination an ill defined mass was palpable in the right rectus region No rigidity or spasm was noted The white blood count was 12 000 The temperature which had been normal now rose to 100.4 degrees The patient appeared to be in partial shock being cyanotic and sweating with cold extremities

A diagnosis of recurrent intestinal obstruction was made and operation was performed that night The patient was found to have a mesenteric thrombosis with gangrene of the bowel for 18 inches The involved segment was resected and an end-to-end anastomosis was performed Following the second operation the bowels moved freely and gas was passed per rectum but the distention

remained. A Levin tube was introduced and continuous suction instituted. As the distention did not respond to treatment the Levin tube was removed and a Cantor tube was passed. This readily emptied the jejunum and a large amount of intestinal contents was aspirated. The distention rapidly disappeared. Suction was maintained for two weeks, electrolyte balance being maintained by intravenous fluid. It was noted during this period that when the tube was clamped for more than four hours the patient complained of fullness and the distention reappeared. Thirty cubic centimeters of barium suspension were injected through the tube to ascertain the cause for this difficulty, since the bowels moved daily and flatus was passed freely per rectum. Figure 263 is an X ray taken at the end of five hours showing definite puddling of the barium just beyond the tip of the tube. A 24 hour film showed the same puddling distal to the end of the tube but a small amount of the barium had passed into the colon. A roentgen diagnosis of partial bowel obstruction was made and surgery advised. At operation a partial obstruction of two separate loops of ileum was found distal to the end

of the tube. One obstruction was at the site of the anastomosis as a result of a constricting band between the bowel and the mesentery and the second obstruction was caused by a loop of bowel becoming adherent to the raw surface left in the posterior peritoneum. After liberation of these bands the patient had an uneventful recovery.

This case exemplifies the exactitude with which the radiologist is able to diagnose a partial obstruction with perfect safety by injecting a barium suspension through the intestinal tube. Clinically this patient would have been considered non-obstructed since his bowels moved daily and he passed gas freely. Removal of the intestinal tube without relieving the partial obstruction might have been serious.

Cas 3

A 55 year old white woman was admitted to the hospital with a history of sudden onset of severe pain in



FIG 263 Barium suspension injected through a Cantor tube to determine the cause of abdominal distention following surgery. A definite puddling of the barium was noted distal to the end of the tube, indicating a high grade partial obstruction. At operation a high grade partial obstruction of two separate loops of ileum was found distal to the end of the tube.



FIG 264 The use of the long intestinal decompression tube and injection of barium to demonstrate the patency of anastomosis following bowel resection. It is apparent from this film that the site of anastomosis is patent, the barium being found in the right colon. The tube can therefore be removed with safety.

the abdomen full, followed shortly by vomiting and distention. Upon examination the abdomen was found to be greatly distended, with an ill defined mass in the right lower quadrant. A barium enema ruled out an obstructive lesion of the colon. The patient was operated upon and a volvulus of the terminal ileum was found with 3 feet of gangrenous bowel. Resection of the involved segment was performed with an end-to-end anastomosis. Following the operation the patient began to vomit and the distention increased. A long intestinal tube was passed and proceeded to the lower jejunum within 19 hours. The distention rapidly subsided and the vomiting ceased. The surgeon was apprehensive as to the patency of the anastomosis because of the questionable blood supply of the portion of the ileum attached to the cecum. For this reason on the seventh day 50 cc of dilute barium were injected through the tube. Following this the tube was clamped for five hours and an X ray was taken (Figure 264). This shows definite passage of the barium into the colon and absence of puddling distal to the end of the tube. The surgeon was advised that the anastomosis was patent and adequate. The intestinal tube was then removed.

This case exemplifies the ease with which the patency of an anastomosis can be demonstrated radiographically and the exactness with which the safe period for removal of the tube can be determined.

Case 4

E. T., a 70 year-old white woman was admitted to the hospital complaining of severe pain in the abdomen. The pain had begun the day prior to admission and was cramping in character and diffuse. It was followed rapidly by distention and vomiting. Upon admission the abdomen was found to be tremendously distended but soft. No mass or spasm was palpable. Definite peristaltic activity was audible. A barium enema revealed a normal colon. A Cantor tube was passed with ease into the ileum and by the fifth day all signs of intestinal distention had disappeared. Fifty cubic centimeters of barium suspension were injected through the tube to determine the cause of the abdominal distention. Figure 265 is an X ray taken five hours after the injection of the barium. It is quite



FIG 265 The use of barium suspension through a Cantor tube to determine the cause of abdominal distention. This figure demonstrates that five hours after the injection of barium there is no mechanical obstruction in the bowel since the barium passes readily through the small bowel.



FIG 266 Acute intestinal obstruction due to volvulus at the terminal ileum caused by a previously placed side-to-side anastomosis. Note the relative absence of intestinal distention, there being only one distended loop of small bowel noted radiologically. In this type of case the radiologic findings may be meager early in the course of the disease.

apparent that there is no mechanical obstruction in the bowel. The barium passed readily into the colon. The intestinal tube was therefore removed. A specific diagnosis as to the cause of the distention was never made.

The radiologic method of determining the continuity of the small bowel was utilized in this case to save the patient from the hazards of an exploratory laparotomy. The barium enema disclosed no lesion of the colon, and the free passage of the barium through the small bowel with the intestinal tube in place assured the surgeon that no mechanical obstruction was present. In the event that a bowel obstruction had been present, the barium suspension could have been suctioned out or flushed out by irrigation through the tube. In that event an accurate localization of the point of obstruction could have been made so that operative manipulation of the bowel would be reduced to a minimum.

It is often difficult to determine clinically whether the case is one of ileus or partial bowel obstruction due to adhesive bands or whether the adhesion of loops of bowel to a granulation area within the peritoneal cavity is the cause of the intestinal distention. The radiologic method can safely and accurately answer these questions.

In the event that an obstructive lesion is found its location in the bowel can be determined fairly accurately. This obviates the necessity of much bowel handling by the surgeon and thus decreases the incidence of postoperative ileus.

The method is simple and safe. In the presence of complete bowel obstruction the barium suspension which is very dilute and small in amount can easily be suctioned or flushed out by irrigation with normal saline through the intestinal decompression tube.

DISTENTION IN THE GASTRO- INTESTINAL TRACT

The phenomenon of intestinal distention is an invariable accompaniment of any interference with the normal passage of intestinal contents from the mouth to the anus. Once there is an interference with the free movement of the intestinal stream some degree of distention is almost certain to follow.

Medical interest in the problem of intestinal distention has been traced back to the time of Hippocrates whose book on the winds or flatuosities is the earliest written work on this subject. In ancient times this condition was given the name morbus ructuosus and all sorts of disorders were ascribed to it.

Although Van Helmont conducted a study of intestinal gases in 1652 and Combalusier in 1747 attempted to give a more restricted significance to the role played by the gases it was not until the end of the 18th century that the indefinite notion of morbus ructuosus was abandoned and interest became centered on the actual composition and origin of the gases. Both human beings and animals were studied and bit by bit the true nature of intestinal gases began to be understood. Studies of human subjects largely criminals examined after death produced a considerable increase in the knowledge of the source and nature of these intestinal gases. Despite the crude methods employed at the time the basis for the chemical knowledge of the subject was really laid down by these early investigations.

SOURCES OF GAS CAUSING DISTENTION

There are three sources of gas capable of causing intestinal distention.

Air Swallowing

Magendie gave us the first clear account of air swallowing in 1813. He reported the case of an army conscript who practiced auto inflation in order to avoid military service. The experimental contributions of Latour, Tappeiner, Schierbeck, Woodyat, Graham and Ylppo all tended to indicate that under normal conditions the basis of gastric gases was atmospheric air. Despite the work of Magendie which indicated that air swallowing was both a common and a perfectly normal physiologic process it was not fully realized that air swallowing under pathologic conditions could give rise to disturbances generally assumed to be due to gases arising within the body. It was not until the publications of Quincke in 1889 and of Bouveret in 1891 that the present viewpoint was given complete expression. Bouveret's term aerophagia was immediately accepted and confirmatory papers quickly followed one another. Although it was readily accepted that air was constantly being swallowed by normal individuals it was not so widely accepted that the gases of the stomach were derived entirely from the atmosphere. The observation that air could be swallowed into the stomach and that such respiratory sucking of air accounted for the free gas in the stomach and intestines of the newborn was first made in 1877.

The fate of atmospheric air that is swallowed and appears in the stomach of the normal individual is well known. Most of the swallowed air escapes through the esophagus by eructation. This is a reflex and occurs when there is an increase in the intragastric pressure. Some of the gas passes

through the pylorus and moves downward into the gastro intestinal tract. A portion of this gas is excreted per rectum and a portion of it is absorbed by the mucosa. In 1931 Magnusson demonstrated that air injected into the stomach would pass through the small bowel to the cecum in approximately 10 minutes and would result in the passage of flatus per rectum in 30 minutes. The fact that oxygen is readily absorbed by the gastric mucosa is an old observation. Hippo showed that oxygen is absorbed by the gastric mucosa at the rate of 12 cc per minute. It is therefore probable that whatever gas is passed into the small intestine at the end of the ordinary period of digestion is composed largely of nitrogen with some carbon dioxide from the stomach. In the fasting stomach whole atmospheric air may pass through the pylorus. Maddock, Bell and Tremaine studied intestinal gases by observations of belching during anesthesia at surgery and during pycnography. The mechanism proposed by these authors to explain the rapid accumulation of gas in the gastro intestinal tract is as follows. Normally the superior esophageal sphincter keeps the esophagus closed. However, with swallowing movements the superior sphincter relaxes and air enters with fluid and food. In the upright position the air collects at the top of the stomach and when more than the usual amount accumulates as with a meal it is belched up.

Excessive amounts of air have been shown to enter the stomach by frequent swallowing and also by the following method which is a variation of this same procedure. The air sucker can consciously relax the superior esophageal sphincter and by attempting to breathe against a closed glottis can aspirate air into the esophagus from whence it passes into the stomach. An aerophagic is a nervous person who unconsciously does the same thing and usually must be convinced of what he is doing to be cured of the habit. The laryngotomized patient can learn esophageal speech with air aspirated into the esophagus using the same method as that of the air sucker. With skill his aspirations occur with almost normal inspiration and are practically unnoticeable. Similarly the reported belcher takes air into the esophagus by the same method as the air sucker and some can

say a few words with the crupted air in the same manner as the esophageal speech patient.

From these observations Maddock, Bell and Tremaine concluded that appreciable volumes of air enter the stomach through repeated belching. There is every reason to believe that many patients under many different conditions may temporarily become air swallows and that the stimulus may be entirely nervous without organic origin although it may be associated with an organic disease or injury. In intestinal obstruction this swallowed atmospheric air has been found to be responsible for 60 to 70 per cent of the gas found in the gastro intestinal tract.

Interchange of Gas Between Blood and Bowel Wall

Not all of the gas found in the gastro intestinal tract is the result of swallowed air. The gaseous interchange with the circulating blood through and to the bowel wall is believed to be responsible for 20 per cent of all gas found in the gastro-intestinal tract in cases of intestinal obstruction. Throughout the gastro intestinal tract a diffusion of the intestinal gases occurs between the blood and tissue gases and those within the lumen of the bowel until there is an equalization of gas pressures on each side of the semipermeable membrane which in this case is the bowel wall. A continuous interchange of gases occurs between the blood and the bowel lumen. The direction of flow and the amount of gas flowing depend upon the partial pressure for that specific gas. When the tension for a gas within the bowel is greater than that of the surroundings the specific gas within the bowel diffuses over to the blood and the tissue. The rate of this process of diffusion is expressed by the absorption coefficient for that gas. This absorption coefficient depends upon the solubility in water of the gas in question. Carbon dioxide is diffused very rapidly while oxygen and especially nitrogen spread very slowly in the tissues. In addition it must be noted that the tissue content of oxygen and carbon dioxide is to a very great extent dependent upon the state of the circulation. Warren demonstrated in 1937 that under experimental conditions of shock the carbon dioxide content of

the tissues rose very rapidly while the oxygen content fell

The laws of pure physics which may be applied to the problem of intestinal gas and diffusion through the tissues are Dalton's Law and Henry's Law. Dalton's law states that in a mixture of gases each gas exerts the pressure it would exert separately. Henry's law states that if a gas is in contact with a fluid the amount of gas absorbed by the fluid is in direct proportion to the partial pressure of the gas. The combination of these laws with the study of diffusion of gases through biologic membranes such as the bowel wall has produced the following equation

$$D = k \frac{a (p^1 - p^2)}{760 d} \frac{m}{m}$$

In this equation D is the amount of air which is diffused through the membrane in a given period of time a is the absorption coefficient $p^1 - p^2$ is the difference in pressure on each side of the semi permeable membrane d is the thickness of the membrane and m is the molecular weight of the gas in question. The absorption coefficient has been determined for all the various gases so that using this equation one can determine with a reasonable degree of accuracy the quantity of air which diffuses in a given period of time through the membrane. The absorption coefficient for nitrogen is 0.012. The absorption coefficient for oxygen is 0.024 and for carbon dioxide 0.34. It is thus apparent that carbon dioxide is the most rapidly taken up and nitrogen the least. Tacke demonstrated in experiments on rabbits that under normal conditions the main portion of all gases formed in or introduced into the gastro intestinal canal is removed by the blood stream and the lungs with only a small portion passing through the anus. The studies of McIver, Redfield and Benedict have demonstrated that oxygen may be absorbed approximately 1.5 times as rapidly as nitrogen and carbon dioxide 160 times as rapidly as nitrogen. It was also demonstrated that this process of diffusion of gases was to a very great extent influenced by the circulation within the bowel wall. Compression of the aorta was shown to suspend resorption of the carbon dioxide introduced into the bowel. From this it should be quite evident

that strangulating types of obstruction or any obstruction in which there is compression of blood vessels would interfere markedly with the ability of the bowel to absorb the intestinal gas present within its lumen.

Action of Intestinal Bacteria on Bowel Content

The action of intestinal bacteria upon the bowel content results in the formation of approximately 10 per cent of all gas within the bowel in intestinal obstruction. The terminal ileum and the right half of the colon have the required factors of stagnation, fluidity and an abundance of intestinal bacteria permitting fermentation and putrefaction. The production of gas from intestinal contents is extremely variable and depends to a great extent upon the composition of the food ingested. Under conditions of restricted food administration or during starvation this source of intestinal gas production is of very slight importance.

COMPOSITION OF GAS WITHIN THE BOWEL

Analysis of the gas found in the gastro intestinal tract shows that nitrogen, oxygen, hydrogen, methane and hydrogen sulfide are present. The proportions of these gases vary widely depending upon the type of food ingested and in cases of intestinal obstruction upon the duration of the obstruction and its level along the gastro intestinal tract.

Many studies have been made of the colonic gases and the composition of intestinal flatus has been reported by various authors. In Fries' analysis of flatus the percentage composition by volume is shown to be as follows: carbon dioxide 10.3 per cent, oxygen 0.7 per cent, methane 29.6 per cent, nitrogen 59.4 per cent. In addition, Fries found that in the healthy adult male single discharges of flatus per rectum vary from 50 to 500 cc with an average of 100 cc and a daily total of approximately 1000 cc. In 1945 as a result of their studies on intestinal gases, Ringsted and Anderson concluded that nitrogen was by far the principal component of the intestinal gas associated with intestinal obstruction. They verified this by finding that flatus from patients with postoperative

through the pylorus and moves downward into the gastrointestinal tract. A portion of this gas is excreted per rectum and a portion of it is absorbed by the mucosa. In 1931 Magnusson demonstrated that air injected into the stomach would pass through the small bowel to the cecum in approximately 10 minutes and would result in the passage of flatus per rectum in 30 minutes. The fact that oxygen is readily absorbed by the gastric mucosa is an old observation. Ylppo showed that oxygen is absorbed by the gastric mucosa at the rate of 12 cc per minute. It is therefore probable that whatever gas is passed into the small intestine at the end of the ordinary period of digestion is composed largely of nitrogen with some carbon dioxide from the stomach. In the fasting stomach whole atmospheric air may pass through the pylorus. Maddock, Bell, and Tremaine studied intestinal gases by observations of belching during anesthesia at surgery and during pyelography. The mechanism proposed by these authors to explain the rapid accumulation of gas in the gastrointestinal tract is as follows. Normally the superior esophageal sphincter keeps the esophagus closed. However, with swallowing movements the superior sphincter relaxes and air enters with fluid and food. In the upright position the air collects at the top of the stomach and when more than the usual amount accumulates as with a meal it is belched up.

Excessive amounts of air have been shown to enter the stomach by frequent swallowing and also by the following method which is a variation of this same procedure. The air sucker can consciously relax the superior esophageal sphincter and by attempting to breathe against a closed glottis can aspirate air into the esophagus from whence it passes into the stomach. An aerophagic is a nervous person who unconsciously does the same thing and usually must be convinced of what he is doing to be cured of the habit. The laryngectomized patient can learn esophageal speech with air aspirated into the esophagus using the same method as that of the air sucker. With skill his aspirations occur with almost normal inspiration and are practically unnoticeable. Similarly the reported belcher takes air into the esophagus by the same method as the air sucker and some can

swallow a few words with the erupted air in the same manner as the esophageal speech patient.

From these observations Maddock, Bell, and Tremaine concluded that appreciable volumes of air enter the stomach through repeated belching. There is every reason to believe that many patients under many different conditions may temporarily become air swallows and that the stimulus may be entirely nervous without organic origin although it may be associated with an organic disease or injury. In intestinal obstruction this swallowed atmospheric air has been found to be responsible for 60 to 70 per cent of the gas found in the gastrointestinal tract.

Interchange of Gas Between Blood and Bowel Wall

Not all of the gas found in the gastrointestinal tract is the result of swallowed air. The gaseous interchange with the circulating blood through and to the bowel wall is believed to be responsible for 20 per cent of all gas found in the gastrointestinal tract in cases of intestinal obstruction. Throughout the gastrointestinal tract a diffusion of the intestinal gases occurs between the blood and tissue gases and those within the lumen of the bowel until there is an equalization of gas pressures on each side of the semipermeable membrane, which in this case is the bowel wall. A continuous interchange of gases occurs between the blood and the bowel lumen. The direction of flow and the amount of gas flowing depend upon the partial pressure for that specific gas. When the tension for a gas within the bowel is greater than that of the surroundings the specific gas within the bowel diffuses over to the blood and the tissue. The rate of this process of diffusion is expressed by the absorption coefficient for that gas. This absorption coefficient depends upon the solubility in water of the gas in question. Carbon dioxide is diffused very rapidly while oxygen and especially nitrogen spread very slowly in the tissues. In addition it must be noted that the tissue content of oxygen and carbon dioxide is to a very great extent dependent upon the state of the circulation. Walgren demonstrated in 1937 that under experimental conditions of shock, the carbon dioxide content of

the tissues rose very rapidly while the oxygen content fell

The laws of pure physics which may be applied to the problem of intestinal gas and diffusion through the tissues are Dalton's Law and Henry's Law. Dalton's law states that in a mixture of gases each gas exerts the pressure it would exert separately. Henry's law states that if a gas is in contact with a fluid the amount of gas absorbed by the fluid is in direct proportion to the partial pressure of the gas. The combination of these laws with the study of diffusion of gases through biologic membranes such as the bowel wall has produced the following equation

$$D = k \frac{a (P^1 - P^2)}{760 d} \frac{1}{m}$$

In this equation D is the amount of air which is diffused through the membrane in a given period of time a is the absorption coefficient $P^1 - P^2$ is the difference in pressure on each side of the semi-permeable membrane d is the thickness of the membrane and m is the molecular weight of the gas in question. The absorption coefficient has been determined for all the various gases so that using this equation one can determine with a reasonable degree of accuracy the quantity of air which diffuses in a given period of time through the membrane. The absorption coefficient for nitrogen is 0.012. The absorption coefficient for oxygen is 0.024 and for carbon dioxide 0.14. It is thus apparent that carbon dioxide is the most rapidly taken up and nitrogen the least. Tacke demonstrated in experiments on rabbits that under normal conditions the main portion of all gases formed in or introduced into the gastro intestinal canal is removed by the blood stream and the lungs with only a small portion passing through the anus. The studies of McIver, Redfield and Benedict have demonstrated that oxygen may be absorbed approximately 13 times as rapidly as nitrogen and carbon dioxide 160 times as rapidly as nitrogen. It was also demonstrated that this process of diffusion of gases was to a very great extent influenced by the circulation within the bowel wall. Compression of the aorta was shown to suspend resorption of the carbon dioxide introduced into the bowel. From this it should be quite evident

that strangulating types of obstruction or any stricture in which there is compression of blood vessels would interfere markedly with the ability of the bowel to absorb the intestinal gas present within its lumen.

Action of Intestinal Bacteria on Bowel Content

The action of intestinal bacteria upon the intestinal content results in the formation of approximately 10 per cent of all gas within the bowel in intestinal obstruction. The terminal ileum and the right side of the colon have the required factors of stationary fluidity and an abundance of intestinal bacteria permitting fermentation and putrefaction. The production of gas from intestinal contents is extremely variable and depends to a great extent upon the composition of the food ingested. Under conditions of restricted food administration, during starvation, this source of intestinal gas production is of very slight importance.

COMPOSITION OF GAS WITHIN THE BOWEL

Analysis of the gas found in the gastro intestinal tract shows that nitrogen, oxygen, hydrogen, methane and hydrogen sulfide are present. The proportions of these gases vary widely depending upon the type of food ingested and in cases of intestinal obstruction upon the duration of the obstruction and its level along the gastrointestinal tract.

Many studies have been made of the composition of intestinal flatus. The composition of intestinal flatus has been reported by various authors. In a series of analyses of flatus, the percentage composition by volume shown to be as follows: carbon dioxide 10.3 per cent, oxygen 0.7 per cent, methane 29.6 per cent, nitrogen 59.4 per cent. In addition, it was found that in the healthy adult male single charges of flatus per rectum vary from 10 to 100 cc with an average of 100 cc and a daily total of approximately 1000 cc. In 1941, as a result of studies on intestinal gases, Ringsted and Aronson concluded that nitrogen was by far the principal component of the intestinal gas mass in cases of intestinal obstruction. They verified this finding that flatus from patients with postoperative

intestinal distention contains 80 to 90 per cent nitrogen. Varying low values were found for the other gases such as carbon dioxide, oxygen, methane, and hydrogen. Since the nitrogen represents the main constituent of the gas in intestinal obstruction distention Ringsted and Anderson believed that it originated almost exclusively from swallowed air. Since nitrogen is absorbed slowly, it is very probable that the spontaneous absorption of this gas through the intestinal wall could not keep pace with the increase in nitrogen caused by swallowing in any case of ileus and the distention therefore becomes aggravated. Walgren on the other hand believes that the most important factor in the production of intestinal distention associated with paralytic ileus is the raised carbon dioxide content of the venous blood and tissues associated with this disease. Of the gases found in the human gastro-intestinal tract hydrogen and methane generally are found in such small amounts that they are of little or no importance in the problem of intestinal distention associated with bowel obstruction.

Kugel analyzed colonic gas and correlated the type of intestinal gas present with the diet of the patient. The following analyses were obtained. On a milk diet after 24 hours the following gases were found: carbon dioxide 16.8 per cent, methane 0.9 per cent, hydrogen 43.9 per cent, and nitrogen 38.4 per cent. On a meat diet after 24 hours the following composition was obtained: carbon dioxide 13.6 per cent, methane 37.4 per cent, hydrogen 3 per cent, nitrogen 46 per cent. On a legume diet the following percentages were obtained: carbon dioxide 34 per cent, methane 44.6 per cent, hydrogen 23 per cent, nitrogen 19.1 per cent. It would thus be apparent that with the patient on a meat diet or a mixed diet the predominant gas is nitrogen. This nitrogen is believed to come from several sources. Some is swallowed with the air while eating or drinking, some is swallowed with the saliva, and some nitrogen is excreted from the blood into the bowel. On a legume diet, however, it is apparent that methane is the predominant gas, and on a diet rich in milk, hydrogen predominates. Both the hydrogen and the methane are combustible gases.

COMPOSITION OF INTESTINAL CONTENT (OTHER THAN GAS) CAUSING DISTENTION

In contradistinction to most cases of early postoperative distention in which the distending element is chiefly swallowed air, in the majority of cases of bowel obstruction the distending element is not only gas but also large amounts of fluid and particulate matter. The fluid content of the distending element in the gastro-intestinal tract may equal or exceed the gaseous. This fluid is derived from three main sources:

1. That liquid ingested by the patient and carried down the gastro-intestinal tract to the area of bowel blockage.
2. Saliva, gastric juice, pancreatic juice, bile, and 3000 cc. of succus entericus which is secreted daily in addition to all other secretions.
3. Fluid from the blood vessels of the bowel wall passing into the bowel lumen.

The only constant of this triad is the fluid secreted by the patient. The greatest source of fluid within the bowel in intestinal obstruction is believed to come from the circulating blood and is characteristically increased by circulatory stasis. The only fraction of fluid controllable by the surgeon is that due to liquid ingested. Although it was formerly thought that intestinal distention decreased the secretion of fluid in the bowel, it has been shown by Fine and his co-workers that the greatest stimulus to fluid transudation within the lumen of the bowel is due to intestinal distention. It has been also shown by Gatch and Buttersby that the amount of transudation is almost directly proportional to the degree of intestinal distention and the degree of circulatory embarrassment. As a result of experimental work on dogs, they concluded that the fluid found in the peritoneal cavity in cases of intestinal distention comes in part from the distended bowel and that it tends to increase as the intra-intestinal pressure increases. Herrin and Meek, in studying the effect of distention in dogs with various types of fistulae, have come to the conclusion that distention is a strong stimulus to intestinal secretion. In this fashion a vicious circle is created.

CAUSE OF INTESTINAL DISTENTION

Distention of the gastrointestinal tract varies greatly in degree and in composition depending upon the cause of the interruption of the intestinal stream, the point in the gastrointestinal tract at which the interruption occurs, and the duration of the obstruction. In general, obstruction to the intestinal stream may be produced in three different ways:

- 1 Mechanical occlusion of the bowel either from without or within the lumen of the bowel, or from within the wall of the bowel.
- 2 Diminution or loss of intestinal peristalsis from any cause.
- 3 Interference with the blood supply of the bowel which may be primary or secondary to any of the preceding forms of interference with the intestinal stream.

Any of these factors may operate independently or in combination to produce intestinal stasis which results in distention. The intestinal distention, although an almost invariable accompaniment of this stasis, does not always occur with bowel obstruction. Obstructions high up in the gastrointestinal tract produce very little distention for the obvious reason that there is very little bowel proximal to the intestinal obstruction to become distended. Naturally, the degree of intestinal distention increases as the point of intestinal blockage moves downward to the anus. As would be expected, the greatest degree of intestinal distention occurs with lesions of the sigmoid colon.

Vitamin Deficiency

Vitamin B deficiency, particularly thiamin chloride, may be responsible for many of the cases of postoperative distention. This observation, originally reported by Monteiro and Ilho, was recently substantiated by Leithauser. The latter reported six cases of adynamic ileus with severe abdominal distention resulting from thiamin chloride deficiency. The distention was severe and of such a degree that a diagnosis of intestinal obstruction was suggested. In these cases the distention was not controlled by mechanical decompression nor by administration of Prostigmin but responded dramatically to the administration of thiamin chloride and vitamin B complex. Monteiro and

Ilho were very enthusiastic about the use of thiamin chloride in the treatment of postoperative intestinal distention. Basing their observations upon 19 cases, they demonstrated the therapeutic effectiveness of vitamin B in the treatment of the postoperative distention, particularly when the distention occurred in individuals with a thiamin deficiency.

This experience suggests that a nutritional deficiency should be suspected and a therapeutic trial of vitamins made in cases of abdominal distention in which the evidence does not justify a positive diagnosis of mechanical obstruction. It suggests also that thiamin chloride should be administered at the time of operation to prevent postoperative distention, especially in patients whose nutritional status is at all questionable or in whom there is any suspicion of liver damage.

Reflex Causes

The reflex cause, such as ileus, which may be associated with overstimulation of the sympathetic nerves, should be mentioned among the causes of intestinal distention. Distention may be provoked by the antiparasymphathetic drugs such as Banthine or Pro Banthine. In addition, overactivity of the sympathetic nervous system, which occurs with pneumonia or lumbar myositis, may also produce reflex intestinal distention. Among the neuromuscular disorders producing intestinal distention, hypoproteinemia frequently causes gas distention of the small bowel. This may be the result of giving large amounts of intravenous saline after abdominal surgery. In those cases in which hypoproteinemia causes intestinal distention, it has been found that edema begins when the blood protein drops to 5.5 per cent. In such cases, the intestine is not affected by Prostigmin. This suggests a lack of acetylcholine in the intestinal wall. In some patients who have hypopotassemia, intestinal distention may appear with normal serum proteins. A loss of potassium may result in intestinal distention when the serum potassium falls below 3.5 mEq per liter and it becomes severe at 2.6 mEq per liter. In such cases, the symptoms are quickly relieved by the administration of potassium.

Disease of the mesentery may affect the nerve fibers to such an extent that localized dilatation of

the intestine results. If a cholinesterase inhibitor such as Prostigmin does not affect the intestine, a deficiency of acetylcholine may be assumed. If an acetylcholine derivative such as Urecholine stimulates the intestine and reduces the dilatation, this suggestion is confirmed. If Urecholine is ineffective, the wall of the intestine will not react to acetylcholine. This indicates organic disease of the bowel wall.

Organic Disease of the Bowel Wall

Amyloidosis, scleroderma, and sprue are unusual organic causes of intestinal distention. In the conditions, contraction of the intestinal wall is not induced by parasympathomimetic drugs. The disease process is inherent in the musculature of the small bowel with the result that contraction cannot occur.

Psychosomatic Causes

In an occasional case no reasonable explanation for the intestinal distention may be found. It has been pointed out on many occasions that profound mental and psychosomatic disturbances may result in functional abdominal distention to such a degree that it may simulate intestinal obstruction. The mechanism by which this occurs has not been fully explored as yet.

EFFECTS OF INTESTINAL DISTENTION

That distention alone is capable of producing many of the symptoms and sequelae of intestinal obstruction has been well demonstrated experimentally by Linc, Kosenfeld, and Gendel. These workers demonstrated that distention without obstruction could produce dangerous effects. The effects of intestinal distention may be divided into the local effects upon the gastro intestinal tract and the general effects upon the individual as a whole.

Local Effects

One local effect of intestinal distention is an impairment of the primary physiologic function of the gastro intestinal tract, nutrition. Intestinal distention produces stasis of the intestinal stream so that little or no food is absorbed. As a result the individual loses his power to assimilate food by

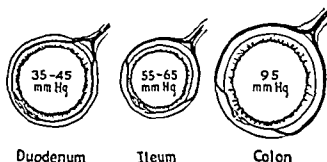


FIG. 267 The point of entrance of the artery into the bowel wall is shown. Notice that as one goes down ward in the gastro intestinal tract the artery remains subserrous for a greater distance along the circumference of the bowel wall and hence it is less likely to be compressed by a distended bowel.

ingestion. Gatch and Battersby have shown experimentally that the power of absorption from the gastro intestinal tract ceases when the intraintestinal pressure equals or exceeds the diastolic blood pressure.

Because the intestinal distention that we are concerned with in intestinal obstruction is the result of the accumulation of gas and fluid in varying proportions, the laws of equalization of pressures do not apply as they would if the distending element were either gas or liquid alone. The admixture of gas and fluid in loops of bowel which are lying in every conceivable plane in the abdomen and are often kinked because of the weight of the fluid within them necessarily decreases the efficiency of any method of suction applied for the purpose of decreasing the distention. These kinks in the bowel are important factors in decreasing the efficiency of gastroduodenal suction drainage. For this reason and because such kinks in the bowel may be maintained by the weight of overlying loops of bowel, the use of the long, intestinal decompression tube is essential if decompression is to be obtained.

Effect of Distention upon Circulation of Bowel Wall. The effect of distention upon the circulation of the bowel wall is variable because of the anatomic differences that exist in different parts of the gastro intestinal tract. Dragstedt, Lang, and Millett have shown that there is considerable variation in the intramural blood supply in the different parts of the gastro intestinal tract. The effect of distention upon the blood supply to

the gastrointestinal tract depends upon the point of entrance of the blood vessels between the inner circular and the outer longitudinal muscles of the bowel. The degree of interference with the circulation is greatest in the upper bowel and least in the colon since the blood vessels of the colon do not pass between the muscle layers until a high point on the antimesenteric edge of the bowel has been reached. In the duodenum the blood vessels pass between the muscle layers almost at once and as one proceeds down the small bowel the point of entrance of the blood vessels tends to more closely approximate the antimesenteric border. Dragstedt showed that the decrease in blood flow which he recorded in percentages of normal flow is most marked in the duodenum and least marked in the colon. This would seem to corroborate the anatomic findings in proving his theory as to the effect of intestinal distention upon the circulation in the bowel wall. Gratch and Battersby do not agree with this explanation however because the impairment of the circulation is evidently due to compression of the bowel capillaries and they cannot see how the points at which the vasa recta pierce the muscularis of the bowel can have any effect on this. The anatomic explanation they favor is that there is a gradual decrease in the thickness of the mucosa from the duodenum to the colon. The submucous coat of the bowel offers most of the resistance to the distention and the mucosa surrounded by it is compressed more by the distention than is the muscularis. Therefore they believe that the effect of distention would be most marked where the mucosa is thickest such as in the duodenum and least marked where the mucosa is thinnest as in the colon.

The blood flow through the intestinal wall depends not only upon the intra intestinal pressure but also upon the diastolic blood pressure. A diastolic blood pressure of 70 mm. of mercury and an intra intestinal pressure of 50 mm. of mercury would provide a fairly good blood flow through the bowel wall. If the diastolic blood pressure drops to 30 mm. of mercury then the blood flow through the bowel wall will not be continuous and may even cease when the blood pressure drops as a result of a bowel obstruction. Gratch, Trusler and Ayers have observed that there is always some

flow of blood which no degree of intra intestinal pressure will stop. They attribute this to a small anastomotic artery at the mesenteric border of the bowel. This explains the maintenance of the flow of blood through the mesentery when the circulation through the intestine is arrested by distention. Stone and Liorer have recorded measurements of intra intestinal pressure varying from 13 to 411 mm. of mercury. Sperling, Paine and Wangensteen have reported the rupture of a distended loop of bowel on its escape from the abdomen. They found by experiment that a piece of fresh human ileum ruptured at a pressure of 210 mm. of mercury. Except for such extremely high pressures the intra intestinal pressure is no measure of the impairment of circulation.

Gratch and Battersby have shown that a pressure high enough to put the wall of the bowel under tension will decrease in the course of a few hours because of dilation of the bowel. In such cases the color of the wall of the bowel and its degree of tensility clearly show whether the pressure is injuring it. They have demonstrated in dogs that an intra intestinal pressure great enough to cause tenseness of the wall of the bowel will slowly decrease as the bowel dilates and becomes flaccid. A tense bowel has a diminished blood flow. When the bowel becomes flaccid however there is sufficient blood flow to keep it alive although it suffers from congestion of blood in its capillaries. In this way dilation protects the bowel against high intra intestinal pressure provided the pressure in the bowel does not increase step by step as does the dilation. Acute and serious injury to the bowel by intra intestinal pressure occurs only when the pressure is near the diastolic blood pressure and persists for several hours. Van Zwalenburg made similar observations on the blood flow in the intestinal wall as affected by varying pressures within the intestinal lumen. He noted that it was quite certain that distention of the bowel interfered with the circulation in its wall and permitted infiltration and effusion to take place into its wall and lumen and into any other open spaces which might come under its influence. The venous circulation was shown to be retarded at comparatively slight pressures. Effusion follows as in all obstructions to venous flow. Since the average venous pressure

varies from 4 to 10 mm of mercury any pressure beyond that would offer some resistance to the return flow of blood. Because of the venous compression there is an impairment of absorption of the intraintestinal gases by way of the blood stream.

McIver, Redfield and Benedict indicated that the small bowel alone is capable of absorbing 200 cc of carbon dioxide per hour, 1100 cc of hydrogen sulfide per hour, 240 cc of oxygen per hour and 125 cc of hydrogen per hour with lesser amounts of other gases. It should be evident that any impairment of the ability to absorb these intestinal gases because of an edematous compression or because of an impairment in capillary circulation to the bowel wall would appreciably increase the amount of intestinal distention.

Burget, Mirtzloff, Suckow and Thornton produced isolated closed loop intestinal obstructions in dogs. They attached loops to peritoneal peritoneum in such a fashion that the loops could be readily tapped with a syringe. Frequent tapings of these loops within the first few weeks demonstrated a considerable amount of intestinal distention. After this period however some dogs required only an occasional tapping to relieve the distention and other dogs required no tapping at all. These animals requiring no relief from distention demonstrated at autopsy a marked hypertrophy of the vascular system of the loop which suggested the part played by the circulation in controlling the intraintestinal gaseous pressures.

It has been amply demonstrated that the degree of distention associated with intestinal obstruction results in a directly proportionate circulatory impairment of the bowel wall. Impaired nutrition to the bowel wall is a result of impaired blood supply occurs when the pressure within the lumen of the bowel exceeds the systolic pressure and it may be followed by necrosis and perforation of the bowel. The check mechanisms previously discussed are such that this occurrence is uncommon. Intestinal distention of a degree found clinically although sufficient to impair the venous return because of the low venous pressure usually will not sufficiently impair the arterial blood supply to produce gangrene and perforation except in far advanced or neglected cases.

Effect of Distention upon Motility Distention of any part of the small bowel causes an inhibition of the small intestine both above as well as below the distention. Youmans, Meek and Herrin showed experimentally that the motility of the jejunum above and below a distended bowel loop had been decreased. In a dog, the distention of the jejunum results in inhibition of all types of movement and decreased tonus of the undistended portion of the jejunum in both directions from the site of the distention. The degree of inhibition depends upon the rapidity with which the pressure in the distending balloon is increased and upon the final pressures obtained. This inhibition of the jejunum is the result of a reflex over the extrinsic nerves by the stimulation of the afferent endings in the jejunum. When the extrinsic nerves are cut an intrinsic and less efficient mechanism for mediating intestinal inhibition during intestinal distention is brought into play. This pathway is supposedly over the intrinsic nerve cells in the bowel wall.

Drigstedt suggested that the decreased oxygen saturation of the blood in the wall of the obstructed bowel interferes with the proper function of the myenteric nerve plexus. He believed that this accounts for the disorganization of peristaltic activity associated with paralytic ileus or ileus bowel obstruction.

Effect of Vitamin Deficiency upon Motility When a vitamin deficiency state does not exist it seems doubtful whether any one vitamin or any group of vitamins has any stimulating effect upon intestinal motility. However it has been well established that a lack of certain elements of vitamin B complex will seriously impair intestinal motility. It has also been shown *in vivo* and *in vitro* that vitamin B₁ inhibits the cholinesterase which in turn frees more acetylcholine to promote intestinal motility. Monteiro and Filho in their studies on the effect of thiamin upon intestinal distention were of the opinion that a deficiency of this vitamin resulted in degenerative changes in the plexus of Auerbach and in the nerve endings of the vagus. From its action in the treatment of intestinal distention they formed a hypothesis that the vitamin B₁ had a healing action upon the cells

and the fibers of the plexus of Auerbach and the vagus.

General Effects

Almost all the body systems undergo some physiologic or functional changes as a result of prolonged intestinal distention. Some systems are affected to a greater degree than others but altogether there is little doubt that intestinal distention in itself is capable of causing death.

Effect upon Circulating Blood Volume
Gatch and Pattersby demonstrated that a considerable loss of fluid occurred from the mesenteric border of the distended bowel. In addition they showed that there is an escape of blood proteins from capillaries in all parts of the body as a result of prolonged intestinal distention. This implies that the distention of the bowel injures the capillary endothelium of the blood vessels and permits the blood proteins to escape. They suggest that this is the result of atrophy caused by the bowel distention. Their experimental work has shown that analysis of the blood after prolonged distention of the bowel reveals that more albumin than globulin is lost to the circulating blood. This indicates that the damage to the capillary endothelium is not as grave as that caused by burns or inflammation since after the injuries the large globulin molecules escape as easily as the smaller albumin molecules.

Fine, Fuchs and Gendel, studying the factors causing death in animals with uncomplicated intestinal obstruction, noted a rapid fall in the volume of the circulating plasma. This extreme loss of plasma occurred as a result of distention of the obstructed small bowel and it continued as long as the distention continued. Evidence was obtained that intravenous injections of plasma in amounts adequate to replace that lost as a result of the obstruction and distention of the small intestine conferred a protective influence sufficient to markedly prolong the life of the animal. This effect in prolonging life was not obtained by the use of equal or larger amounts of physiologic sodium chloride. Since the distention is responsible for the loss of plasma, decompression should be accompanied by a retardation of the loss or even by a gain in plasma volume. Clinically, this effect upon the

plasma volume is not noted in the early stages of the obstruction because of the various checks and balances brought into play by the body. When distention develops spontaneously, the tension produced by the intestinal gas when it first enters the bowel is usually not sustained. This is due to absorption of the gas by the blood stream, relaxation of the intestinal muscle tonus, spatial readjustments of adjacent organs and the relaxation of the abdominal wall. The flexibility of these accommodating mechanisms tends to keep the intraluminal pressure from reaching pathologic levels. In addition, a patent pylorus permits the regurgitation of intestinal gas to some extent. Therefore it is only when these adjustments fail that there will be a critical loss of plasma volume such as that noted in experimental animals. In addition to these protective mechanisms, Gatch and Pattersby have demonstrated that if the intraluminal pressure does not keep pace step by step the bowel wall will respond to the increased intraluminal pressure by dilating in an effort to protect itself against injury.

Effect upon the Circulatory System
Associated with a prolonged high degree of intestinal distention, a circulatory impairment develops. This is brought about by several mechanisms. One of these is the limitation of diaphragmatic excursion due to the pushing upward of the diaphragm into the thorax by the distended loops of bowel. The decreased negative pressure in the chest results in a loss of sucking power into the superior vena cava and a resultant congestion of the areas which empty into it. In addition, the abdominal distention by pressure upon the inferior vena cava causes an impaired venous return and consequently a loss of serum into the peritoneal cavity. This is further increased when the patient is put up into a Fowler's position. In this position the flexion of the thighs tends to decrease the venous return to the femoral vein which is further decreased by the increased intra-abdominal pressure pushing upon the iliac vein. Both methods of venous stagnation may produce considerable fluid loss to the circulation of the patient with an obstructed bowel.

Frimann Dahl demonstrated that restricted diaphragmatic movement results in a delay in the portal venous flow. It was also demonstrated that

the flow of blood into the portal vein decreases progressively with an increase in the intra-abdominal pressure caused by a progressively increasing intra-intestinal pressure. Usadel noted a decreased portal blood flow associated with the decreased intestinal motility found in paralytic ileus.

Venous stasis inevitably results from intestinal distention. As a result of this venous stasis tissue anoxia occurs. This is inevitable when one considers the fact that the entire portal venous circulation becomes stagnant as a result of the pressure of the distended bowel upon the veins in the bowel wall. Sperling showed that a sustained intraluminal pressure of 17 cm. of water is capable of injuring the bowel wall through venous stasis. With venous stasis not only is nutrition impaired because of impaired absorption but in addition, the bowel wall becomes permeable to bacteria. Mahoney and Sherman have shown that placing succinylsulfathiazole into closed loops of bowel greatly prolonged the survival rate. This strongly suggests that bacterial products may be a factor in preventing intestinal distention. A further discussion of this point appears in Chapter 25.

Noor Robb and Jacobson studied the circulatory disturbances produced by acute intestinal distention in rabbits. Direct observations of the blood vessels in the bowel wall of these rabbits revealed that there was a demonstrable impairment of the circulation at a pressure of 30 to 40 mm. of mercury. At these pressures, however, deflation resulted in complete recovery of the circulation. Distending pressures of 50 mm. of mercury and greater resulted in marked impairment of circulation with irreversible changes in the circulation of the bowel wall. The changes include permanent interruption of many of the smaller vessels, interstitial hemorrhages and pronounced intravascular sludging of the red blood cells. These observations indicate that early and rapid intestinal decompression is highly desirable in all cases of obstruction.

In addition to its effect upon the blood vessels of the circulatory system the work of Gorelik suggests that the heart itself may become impaired as a result of intestinal distention. In a series of experimental animals in which the anterior descending or the circumflex branch of the

left coronary artery had been ligated Gorelik demonstrated that distention of the colon with air regularly induced fatal coronary insufficiency. Subepicardial hemorrhages were produced particularly in the area of transition between normal myocardium and the infarction. In addition myocardial hemorrhages were found at some distance from the infarct. The explanation which Gorelik proposed for this was the finding that a distended colon markedly elevates and distorts the diaphragm and so tilts the heart that the apex points superiorly thus compromising the coronary flow. In addition to those animals on which partial ligation of the coronary arteries was performed it was also shown that inflation of the colon caused death in 60 normal experimental animals.

Effect upon the Hematopoietic System. It has long been known that serious disturbances may occur in the hematopoietic system as a result of disease affecting the integrity of the gastrointestinal tract. I have first recognized the relationship between microcytic anemia resembling pernicious anemia and intestinal strictures. In the next 30 years many established cases were collected and reported by various authors. In all the reported cases the basic pathology was a stricture along the gastrointestinal tract or a blind loop with stricture without any interference to the passage of intestinal contents along the gastrointestinal tract. In no case had more than 60 cm. of intestine been resected. Since it has been shown that much more extensive resection than this is required to produce anemia both in experimental animals and in humans the consensus at present is that intestinal stagnation is the cause of the microcytic anemia which develops in these cases. In many of the cases reported excision of the stricture and end to end anastomosis completely cured the microcytic anemia. The first case in which excision of a stricture cured the anemia was reported by Syderhelm in 1922. Since then many similar cases have been reported so that a cause and effect relationship has been established. There is every reason to believe that the microcytic anemia in such patients is not related to pernicious anemia but is probably related to a hepatic deficiency. Sprong, Pollock and Mack are of the opinion that as far as the pathogenesis of the stricture type of anemia

is concerned it is quite probable that the etiologic factor is due to stagnation of intestinal contents. They believe that compounds of stagnant intestinal contents act by interfering with the formation of the absorption or the utilization of materials required for normal erythropoiesis.

Demidova demonstrated in a study of the white blood cell count in cases of acute ileus that in the group in which toxic obstruction was noted in the small bowel leukocytosis was soon replaced by a leukopenia. In addition he noted that the duration and level of the obstruction were important factors in determining the type of degenerative change which occurred in the hematopoietic system. The degenerative blood picture may also occur with distention of the small bowel as well as distention of the large bowel and with mechanical obstruction as well as with paralytic ileus. It may also occur without any evidence of any complicating infection or necrosis. These findings confirm the fact that abdominal distention *per se* may be the cause of the degenerative changes shown by the blood picture. The actual mechanism must be an inhibition of the hematopoietic function of the bone marrow by some toxic substance apparently nonbacterial in origin and possibly absorbed from the distended intestine. The effect of this hypothetical substance on leukopoiesis is purely inhibitory and is not due to overstimulation such as is supposed to occur at times because of overwhelming infection. Kraufman and vom Saal have reported on the white blood counts of patients with various intra abdominal diseases. They found that a marked neutrophilic shift to the left may occur in all types of acute peritoneal irritation even without infection. Van Duyn also noted a degenerative blood picture in distention of the small bowel as well as in obstruction of the colon. The same picture occurred regardless of whether the distention was paralytic or mechanical and in the absence of any evidence of infection. These findings confirmed the fact that intestinal distention in itself was the cause of the degenerative blood picture. Whatever may ultimately prove to be the cause of the marked neutrophilic shift to the left whether it be intestinal distention peritoneal irritation or some other factor its importance lies in the fact that it can occur and must not be con-

sidered as being due to the development of infection. This pure degenerative blood picture described by Van Duyn as it occurs in intestinal distention is characterized by low to leukopenic white blood cell counts normal to neutropenic neutrophil percentages absence of myelocytes normal to increased percentages of lymphocytes and monocytes and the presence of eosinophils.

Effect upon Renal Function. It has long been known that there is a rise in the non protein nitrogen in late cases of intestinal obstruction. Tileston and Comfort as early as 1914 reported high non protein nitrogen in the blood of experimental animals with high intestinal obstruction. In 1919 McQuarrie and Whipple studied the influence of intestinal obstruction upon the renal function. They noted that there was a definite impairment of renal excretory function associated with intestinal obstruction. This functional depression was roughly proportional to the degree of obstruction. They also noted a great increase in the non protein nitrogen of the blood which was observed in acute intestinal obstruction. This had hitherto been ascribed to an increased rate of protein destruction. It was now considered to be partially due to retention of these products released from the injured cell protein. It was suggested that there was a possibility that the impaired renal function was due to the direct action of a toxic substance from the gastrointestinal tract acting upon the renal epithelium. This toxic substance was supposedly formed within the lumen of the bowel.

In high intestinal obstruction vomiting is a very early symptom. In a short period of time if not treated it results in the development of dehydration due to fluid loss and alkalosis from chloride loss. This eventually results in renal failure nitrogen retention and death.

Effect upon Blood Pressure and Respiration. Crowley has described the effect of distention upon respiration and blood pressure exclusive of the interference with diaphragmatic excursion. Study in experimental animals has shown that excessive intestinal distention suddenly induced is capable of producing marked variations in blood pressure and respiration even when the animals are anesthetized. These changes persist as long as the intestinal distention which irritated them con-

times. Crowley is of the opinion that these respiratory and blood pressure changes are the result of some obscure reflexes from the bowel wall. The stimuli to afferent endings in the bowel wall relay impulses via the splanchnic nerves to the respiratory and blood pressure centers in the medulla. From these efferent impulses are sent out by way of the autonomic nervous system which sends communicating rami through the cerebrospinal nerves to produce both respiratory and blood pressure changes. These changes are of a characteristic pattern and are accompanied by synchronous fluctuations in the blood pressure. Crowley suggests that some of the shocklike syndromes that occur with bowel obstruction may be caused in this way. Clinically the rapid advent of shock phenomenon in any patient with a strangulating obstruction or one in whom the mesentery is twisted tends to lend corroborative support to this idea. However there is insufficient experimental corroboration to make this more than a theory as yet.

Pain and Nervous Exhaustion Due to Distention. Intestinal pain is one of the cardinal symptoms in distention from bowel obstruction in creases. When the intestinal stasis is due to paralytic ileus the element of pain may be absent but the exhaustion is a result of the infection or the etiologic factor responsible for the ileus then replaces pain in importance. With bowel obstruction intestinal colic is severe and almost continuous. The bowel is generally insensitive to ordinary pain producing stimuli such as cutting, burning and pinching. However excessive degrees of increased intraluminal pressure result in pain which is characteristically cramplike with periodic increases and disappearances depending upon the peristaltic activity. The intensity of the pain is roughly proportional to the amount of distention and the degree of peristaltic activity. When the upper jejunum is the site of the obstruction the pain is felt in the upper abdomen. When the obstructive process is in the ileum the pain is felt down below the umbilicus. In such cases rest is obtained with great difficulty because of the severity of the abdominal pain. The intestinal contractions produced in an effort to overcome the blockage of the intestinal stream are apt to be very severe and will remain so until the obstruction is

relieved or the bowel so distended that it becomes atonic. In strangulating types of obstruction the peristaltic waves are so violent that the pain may be almost unbearable. In late cases this may be complicated by the development of pain due to peritonitis. On the other hand the pain may be the result of peritoneal irritation due to peritonitis and the intestinal distention may be on a paralytic basis. In any event long and continuous pain may result in a deterioration of the physical condition of the patient who is already dehydrated and taxed with a primary lesion producing the obstruction. In some cases the pain stimuli from a strangulated bowel may be so severe that they produce a certain degree of shock phenomenon. Physical exhaustion or shock resulting in death may occur.

Role of Distention in Vomiting. The vomiting associated with any interruption of the intestinal stream is directly related to the distention and may be caused by two distinct factors. On the one hand vomiting may be of reflex origin and on the other the vomiting may be due to the mechanical obstruction which prevents the normal passage of intestinal contents.

Best and Taylor describe reflex vomiting as being the result of the stimulation of afferent receptors in the bowel wall due to intestinal distention. The impulses from these receptors in the bowel wall are relayed centrally by splanchnic afferent fibers to the vomiting center in the medulla. Stimulation of this center results in efferent impulses being sent out from the center via autonomic nerve fibers and by means of rami communicantes to cerebrospinal nerves producing the vomiting. As long as the impulses continue to be carried from the bowel wall to the medulla the vomiting continues and is only relieved by a release in the intestinal distention. In addition to the reflex origin of the vomiting associated with intestinal distention there is a definite mechanical factor. It is quite obvious that any interference with the normal downward movement of the intestinal stream must result in intestinal stasis. Stasis of the intestinal stream results in intestinal distention varying in degree with the point at which the interference occurs. The higher the obstruction the smaller the distention, whereas the lower the obstruction the greater the distention. Regardless of the degree of

distention intestinal stasis always occurs. With intestinal stasis vomiting may occur in one of two different fashions. We have been under the impression that a reversal of the peristaltic waves was possible and was responsible for the vomiting of liquid intestinal contents created by the intestinal stasis. Johnston has advanced the hypothesis that reverse peristalsis never occurs, but that the vomiting is due to a reversal in the direction of flow of the intestinal contents by a sort of reflux mechanism. He suggests that it is brought about by the contractions of the bowel upon the intestinal contents forcing this liquid material against the point of blockage. Since the intestinal content cannot get through it rebounds and creates a reversal of the current thus producing vomiting. Regardless of which mechanism one accepts the fact remains that any interference with the normal downward movement of the intestinal stream results in a reversal of this stream causing vomiting.

Dehydration. Dehydration one of the most important effects of intestinal distention can not be controlled adequately because of our knowledge of fluid balance. The development and the degree of dehydration depend upon the level at which the blockage of the intestinal stream occurs. The higher the obstruction the more rapid the onset and the more severe the degree of dehydration whereas the lower the obstruction the slower the onset of dehydration.

The following sequence of events formerly was considered by most surgeons to cause dehydration. With interruption to the intestinal stream retained contents. Gas and fluid accumulate in the obstructed segments of bowel and involve more segments proximal to the point of obstruction. The bowel becomes more and more distended with a resultant increase in intra intestinal pressure. With the increase in intra intestinal pressure there is an increased transudation of fluid into the bowel wall as a result of vascular compression. With this loss of fluid from the blood vessels there is a diminution in circulating blood volume. This causes a loss of plasma producing a hemoconcentration. There is also a loss of chlorides which varies in degree the higher the gastro intestinal tract is ob-

structed. High obstructions have the greatest chloride loss. Since all the blood volume must be maintained for adequate nutrition fluid is drawn into the circulation from the tissues and water depots of the body to replace that lost into the bowel. The tissue fluid is depleted very soon with the resultant development of the dehydration phenomenon.

The extent to which the gastro intestinal distention is due to the presence of an excessive amount of fluid and particulate matter is not fully realized. Under normal conditions the intestinal mucosa absorbs all or most of the water presented to it so that the content of the small bowel is quite liquid. The ingestion of large amounts of water normally will not result in liquid stools. Excess water ingested is absorbed by the small bowel. Even in the presence of edema large amounts of water may be absorbed from the small bowel. As a result of intestinal distention not only is there a decrease in the absorption of the fluid within the bowel but there is also a stimulus for increased secretion. Consequently the fluid within the gastro-intestinal tract becomes greatly increased because of the intestinal distention and dehydration is produced. It has been shown that the quantity of fluid present within the gastro-intestinal tract in any case of intestinal obstruction is almost directly proportional to the degree of distention. In addition to the marked increase in secretion within the bowel lumen resulting from distention Kim demonstrated that in high obstruction gastric secretion was markedly increased. Thus increased gastric secretion is associated with an increase in chloride secretion. As a result there is an accumulation of gas fluid chlorides and potassium in the fluid within the bowel. All this is lost to the body by diminished absorption as well as because of vomiting.

A loss of chlorides either into the bowel or by vomiting is compensated for by a retention of sodium in order to maintain the proper hydrogen ion concentration. As the loss of chlorides continues insufficient sodium is available to compensate for the chloride loss and as a result sodium must be excreted by the kidneys in order to maintain the proper acid base balance. Since sodium is responsible for water retention in the tis-

sue a loss of sodium through the kidneys carries some water with it. This causes a further water loss thus increasing the dehydration. If this process is not corrected the interstitial fluid reserve becomes exhausted causing the cellular water to be lost. This is very dangerous because of the small margin of safety permitted in the loss of intracellular fluid. If continued cellular death occurs.

In contradistinction to the views held above the observations of Fine and his associates indicate that bowel distention maintained for many hours causes marked concentration of the blood. They believe that this cannot be explained by dehydration or by intraperitoneal loss of fluid. Gatch and Battersby suggest that the hemoconcentration which occurs with intestinal distention may be due to the injury to the capillary endothelium throughout the body as a result of the slowly developing a phoria caused by the intestinal distention. As a result of the capillary damage throughout the body they believe that there is a loss of albumin and to a lesser extent globulin.

EFFECT OF INTESTINAL GASES UPON BALLOONS OF INTESTINAL DECOM- PRESSION TUBES

Physicists have known for over 100 years that rubber membranes were permeable to gases. This fact was first pointed out by Mitchell in 1831. He noted at that time that the rate of permeability varied with different gases used. Concomitant with the tremendous strides made in the development of synthetic rubbers it was noted that the different rubbers behaved differently with each specific gas; that is it was soon noted that the permeability of each type of synthetic rubber differed for each kind of gas. Extensive studies were carried on throughout the world with the final result that the permeability of each type of rubber has been charted for each specific gas under many conditions of temperature and pressure.

Experimental Studies

The studies of numerous workers have demonstrated that the behavior of gases in rubber is similar to that of gases in organic liquids. As a result of this observation rubber may be considered as an organic liquid of high molecular

weight. Thus when a gas is brought into contact with a rubber membrane it goes into solution in the rubber on the one side of the membrane and then emerges on the other side by evaporation. It is quite important to note that this process of permeation involves two separate and distinct factors: first the solubility of the gas into the rubber and second the diffusion of the gas through the rubber and its evaporation on the other side of the membrane. In studying this phenomenon it was soon noted that the permeability of any specific gas through a rubber membrane is independent of any other gas on the other side of the membrane. In addition the law of diffusion of gases applies to the passage of the gas through the rubber membrane—namely any specific gas tends to pass from an area of increased pressure into one of decreased pressure for that specific gas until equal pressures of that gas are obtained on both sides of the rubber membrane. In addition the presence of the other gases on the one side of the rubber membrane has no effect upon the permeability of the rubber to any one specific gas.

The results of extensive research were finally formulated into two laws:

1. Henry's law, which postulates that the gas first dissolves in the rubber to a degree which is in proportion to the pressure.
2. Fick's law, which postulates that the gas dissolved in the rubber diffuses in the rubber toward that portion where the gas is present in a lower concentration and finally evaporates out of the rubber.

Of all the gases found in the gastrointestinal tract in cases of intestinal distention carbon dioxide and hydrogen sulfide are apt to be the most troublesome for the intubator. The reason for this is that these gases are highly permeable to the natural rubber found in the balloons of the long intestinal decompression tubes in use today. In addition the concentration of the gases within the bowel is greater than their concentration within the balloon of the intestinal decompression tube. It should be quite evident that although we try to express all the atmospheric air from the balloon of the intestinal decompression tube before inserting it nevertheless some atmospheric air remains within the balloon. From the preceding discussion

TABLE I

The permeability Q (in 10^{-8} cm³ cm⁻¹ atm⁻¹ sec⁻¹) and the diffusivity D (in 10^{-7} cm² sec⁻¹) of gas in various elastomers

Tem °C	H ₂			O ₂			N ₂			CO			CH ₄		He
	Q	$D \approx \frac{Q}{\rho}$	$D \approx \frac{Q}{h}$	Q	$D \approx \frac{Q}{\rho}$	$D \approx \frac{Q}{h}$	Q	$D \approx \frac{Q}{\rho}$	$D \approx \frac{Q}{h}$	Q	$D \approx \frac{Q}{\rho}$	$D \approx \frac{Q}{h}$	Q	$D \approx \frac{Q}{h}$	Q
Natural rubber															
17	28	73	10	12	12.5	12	4.1	8.0	8.3	72	6.7	7.1	12.5	8.8	16.5
25	33	103	100	18	17.5	18	6.6	11.5	12.5	102	10.5	11	22		23
35	58.5	140	140	28.5	27	28	11.0	20	21	145	17	18.5	36		33
43	77	185	180	33	37	33	16	28	29	185	25	26	50	21	44
50	97	220	230	43.5	49	42	22.5	37	40	220	32	34	64		
Buna S															
17	22.5	80		9.0	9.6		3.0	7.2		71	6.8		10.5		13
25	30.5	90	85	13	14	14	4.8	10	10	94	10	11	16		17.5
35	44	115		20	20		7.8	14.5		130	15.5		26		25
43	59.5	175	150	27.5	28	30	11.5	21	23	165	23	24	35		33
50	74	200		34.5	34		14.5	28		195	29		43		42
Terbanan															
17	7.6	31		2.0	2.4		0.50	1.45		15	1.0		1.3		5.8
25	11.5	42	41	3.2	3.6	4.0	0.89	2.3	2.5	23	1.7	1.8	2.4		8.7
35	17.5	64		5.3	6.3		1.65	4.1		37	3.1		4.7		12.5
43	25.5	86	87	7.7	9.1	9.6	2.5	6.2	6.3	52	4.7	5.3	7.0		16.5
50	31.5	110		10.5	13		3.7	8.6		66	7.0		10.1		21
Necylene G															
17	6.8	29		1.75	2.5		0.53	1.55		12.4	1.3		1.4		
25	10.3	38	37	3.0	3.8	4.0	0.89	2.4	2.5	19.5	2.3	2.4	2.5		
35	16	56		5.1	6.2		1.65	4.4		31	4.2		4.8		
43	23	74	72	7.7	10.0	10.0	2.55	7.2	6.7	43.5	6.8	6.8	7.2		
50	28.5	94		10.1	13		3.55	9.4		56.5	9.1		9.8		
Ogpanol H 200															
17	3.0	10		0.84	0.50		0.11	0.27		2.3	0.32		0.2		3.8
25	4.9	14	14	0.90	0.78	0.84	0.22	0.43	0.42	3.8	0.54	0.55	0.55		5.6
35	8.3	21		1.6	1.55		0.44	0.84		6.6	1.05		1.1		8.5
43	12	31	30	2.6	2.4	2.5	0.78	1.5	1.4	10.0	1.8	1.8	1.9		12
50	16.5	38		3.7	3.4		1.15	2.1		14	2.5		2.9		15.5
Buna bene rubber															
17	23	75		10.1	11		3.4	8.1		80	7.6				
25	32	96		14.5	15		4.9	11		105	10.5				
35	46	125		21	22		7.8	16		140	16				
43	60	160		28	30		11	22		175	22				
50	77	180		37	37		14.5	29		200	28				
Methyl rubber															
17	9.0	27		0.93	0.88		0.20	0.46		3.0	0.36		—		8.1
25	13	39		1.6	1.4		0.36	0.79		5.7	0.63		0.60		11
35	20	61		3.1	2.5		0.8	1.7		10.5	1.3		1.45		16
43	29	80		5.0	4.1		1.3	2.8		17	2.2		2.7		22
50	38	105		7.1	6.1		2.2	4.1		24	3.6		4.4		27
Mipolam MP															
17	2.9	15		0.41	0.90					2.3	0.23				
25	4.4	20		0.70	1.3		0.2			4.0	0.44				
35	7.3	30		1.4	2.4		0.4			7.6	0.83				
43	10.5	39		2.1	3.9		0.7			11.5	1.4				
50	14	0		3.2	5.2					15	1.9				
Thokol B															
17	0.71	7.8		0.11						1.3	0.43				
25	1.2	10.5		0.22						2.4	0.81				
35	2.2	18		0.49						4.8	1.7				
43	3.3	26		0.85						7.7	2.8				
50	4.6	35		1.3						11.0	4.0				

following the law of diffusion of gases one would expect the carbon dioxide to diffuse through the wall of the balloon into its lumen distending it until the pressure of the carbon dioxide within the balloon equaled its pressure around it i.e. in the intestinal tract. The same mechanism would of course apply to the other gases found in the gastro intestinal tract. However as noted previously the other gases except hydrogen sulfide are not nearly as readily diffusible through a rubber membrane.

Cantor Phelps and Esling experimentally studied the effect of intestinal gases upon the bal-

loons of intestinal decompression tubes. The gases used in these experimental studies were carbon dioxide and hydrogen sulfide because as noted these are the most diffusible through a rubber membrane and because their concentration within the bowel increases with the duration of the bowel obstruction. The gases were tested with all the commonly used types of intestinal decompression tubes. The apparatus used to test the effect of the gases upon the balloons of the intestinal decompression tubes was a very simple yet effective one. Figure 268 demonstrates a gas cylinder with a gauge capable of permitting the gas to enter a pressure chamber *P* at a definite rate to compensate for the loss of gas through the rubber connections and the incomplete gas tightness of our equipment. The pressure chamber *P* was then connected to a mercury manometer *m* which measures the pressure within the chamber correctly to the millimeter of mercury. After much experimentation we were able to set our flow of gas into the chamber at such a rate as to keep the pressure of the gas in the chamber fairly constant within narrow limits. In this way we were able to subject our tube heads to varying degrees of pressure at room temperature. In order to insure that the chamber was filled with pure gas at the beginning of each experiment we would place the tube heads within the chamber place the lid upon the pressure chamber and then using the blade of a thin knife under the lid we would turn on a full flow of the pure gas into the chamber at high pressure. If this was done for several minutes the atmospheric air present in the chamber was forced out through the chink made by the blade of the pen knife. At the end of several minutes the gas pressure going into the pressure chamber was reduced and the knife blade quickly removed. Our pressure chamber now contained a pure gas from the cylinder. We now set the flow of gas into the chamber at a rate which would keep the pressure within the chamber at any desired level.

The volume of the gas within the balloons was determined by the water displacement method. By immersing our balloons into a measured amount of water in a measuring flask of 200 cc capacity we were able to determine rapidly and simply the amount of gas within the balloon. The value of-

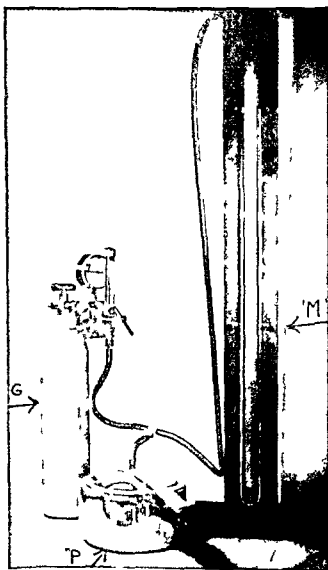


Fig. 268. Apparatus for testing the balloon. *P* is a pressure chamber into which the balloons are placed. *M* is a manometer measuring the gas pressure within the gas chamber. *G* is gas used in experiment.

tained for each balloon before being put into the pressure chamber (with the balloon empty) when subtracted from the value obtained after removal of the balloon from the pressure chamber would be the amount of gas taken up by the balloon in cubic centimeters.

In experimenting with all three types of intestinal decompression tubes in common use today we noted first that the balloons were all of latex rubber so that the diffusibility of any specific gas through this specific rubber should be the same under the same conditions of temperature and pressure. However, on measuring the thickness of the walls of the balloons used with the Miller Abbott and Harris tubes we found them to be 0.006 mm. thick. The thickness of the wall of the Cantor tube balloon ranged from 0.024 mm. at its middle to 0.040 mm. at the base and neck. In addition the Cantor tube and the Miller Abbott tube both presented balloons about 2½ inches long with the diameter of the Miller Abbott tube balloon somewhat greater than the balloon of the Cantor tube. The balloon of the Harris tube, however, is 6 inches long and of the same diameter as the Miller Abbott. The formula quoted by van Amerongen for the quantity of gas permeating through a rubber membrane is as follows:

$$q = Dh \frac{(P^1 - P^2)}{d} t$$

Since d represents the thickness of the rubber membrane it would be evident that the balloons of the Miller Abbott and Harris tubes would be expected to take up more gas than the balloon of the Cantor tube which is four times as thick. Moreover the Harris tube having a balloon 6 inches long would be expected to take up far more gas than any other tube in use today because the value of h which is the area of the rubber membrane is more than double that of either the Miller Abbott or Cantor tube. In the equation quoted above q is the quantity of gas permeating through the specific rubber, D is the diffusibility of each specific gas through each specific type of rubber membrane (this value has been determined for all types of rubber and all types of gas), h is the solubility of the gas in the specific type of rubber membrane (this value has been determined for all types of rubber and all types of gas which

might be found in the gastrointestinal tract), $P^1 - P^2$ is the pressure of the specific gas on each side of the rubber membrane, and t is the time for which the rubber membrane is exposed to the gas.

All the tube heads were then tested under varying conditions to check the applicability of the formula quoted by van Amerongen to clinical medicine. To note the effects of high pressures upon the balloons of intestinal tubes we placed the sealed empty balloons into the chamber and raised the pressure of the carbon dioxide to 180 mm. of mercury for 24 hours. At the end of this period of time we found 10 cc. of gas in the balloons. This rapid observation informed us that gas does enter the balloons. We then proceeded systematically to study these balloons. The following experiments were performed and the results were as indicated:

1 *Experiment 1* To determine whether the silk tie in any way influenced the permeability of the balloon by injuring the rubber.

Result It was found that the silk tie did not injure the rubber and had no influence upon the gas taken up by the balloon.

2 *Experiment 2* To determine the speed with which the carbon dioxide would diffuse out of the balloons when exposed to air.

Result It was found that in 45 minutes the balloons lost one third of the gas within them and at the end of 24 hours the balloons had lost all their gas.

3 *Experiment 3* To determine the effect of a carbon dioxide pressure of 15 mm. of mercury upon balloons exposed for 10 days. New and used balloons were used in this experiment to determine whether soaking the balloon in intestinal contents would affect its permeability.

Result Soaking of the balloon in intestinal contents did not have any effect upon its permeability. Mercury by separating the walls of the balloon created a cavity into which the gas could permeate. If the walls of the balloons were moistened from within so that they stuck together no gas would enter the balloon cavity. The length of time that the mercury remains within the balloon

does not have any effect upon its permeability. If a small opening is made in the balloon such as by the application of a loose tie or puncture of the balloon little or no gas will remain within the balloon.

- 4 *Experiment 4* To determine whether the latex rubber of the Miller Abbott and Harris tubes with a thickness of 0.006 mm. was so highly permeable to carbon dioxide that it could not retain it or whether the walls of the balloon in Experiment 3 were adherent and thus prevented the intake of carbon dioxide.

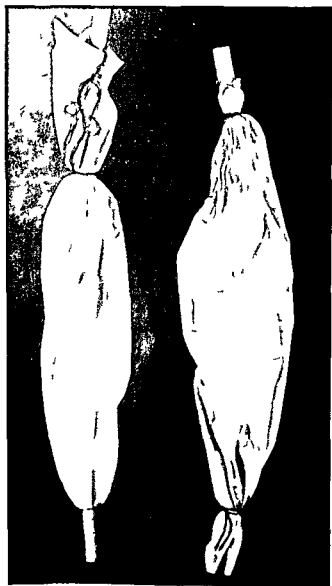


FIG. 269. Balloons of Harris tubes. Left: Balloon containing mercury. Right: Balloon containing no mercury. Note that both balloons contain gas.



FIG. 270. Balloon of Miller Abbott tube. Note gas in balloon.

Result. This experiment showed conclusively that the carbon dioxide gas does diffuse through the balloons of the Miller Abbott and Harris tubes. It also demonstrated that this occurs when the walls of the balloons were kept apart by the shaft of the tube which traverses the balloons. In addition the presence of an inert substance such as mercury which further separates the walls of the balloon permits a greater amount of carbon dioxide to enter the balloons.

- 5 *Experiment 5* To determine whether exposure of the balloons to a pressure of carbon dioxide measuring 100 mm. of mercury for 20 minutes would increase the amount of gas taken up by the balloon.

Result. No carbon dioxide entered the balloon. This indicates that carbon dioxide diffuses into the balloons at such a rate that no increase in the carbon dioxide would be noted at this pressure in a 20 minute period.



FIG 271 Balloon of Harris tube containing glass beads. Note the large amount of gas in the balloon. This gas was then removed and the tube retied. This perfused balloon when subjected to 55 mm pressure of carbon dioxide then took up an equal amount of carbon dioxide gas.

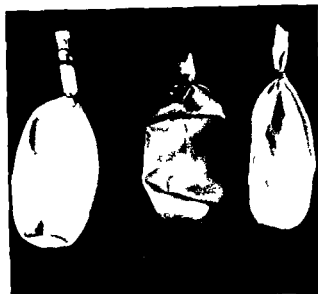


FIG 272 Balloons of Cantor tube. Left Empty balloon. Center Balloon containing mercury this took up little gas. Right Balloon containing glass beads this balloon took up 17 cc of gas.

as an inert mass keeping the walls of the balloon apart thus creating a cavity into which carbon dioxide could permeate. The markedly greater amount of carbon dioxide that diffused into the balloons containing the glass beads as compared with the balloons containing the mercury is understandable when one considers that by the use of glass beads the walls of the balloon are better separated and the small spaces between the surfaces of the beads do not permit the walls of the balloon to rest smoothly upon their surfaces. It becomes evident that anything that will effectively separate the walls of the balloons will result in an increased intake of carbon dioxide. Even with the Miller Abbott and Harris balloons empty the presence of the shaft of the tube passing through these balloons effectively creates a cavity for the intake and storage of carbon dioxide.

- 7 *Experiment 7* To determine whether thorough perfusion of the balloons with carbon dioxide until their walls were saturated with this gas would prevent the intake of carbon dioxide.

Result This experiment conclusively demonstrated that perfusing the balloons

- 6 *Experiment 6* To determine whether the mercury caused more carbon dioxide to enter the balloon because of some special effect within the balloon or whether it was merely the inert mass of mercury separating the walls of the balloon which resulted in the increased amount of gas intake as compared with the empty balloons. For this purpose glass beads were placed in one half of the balloons and mercury in the other half.

Result Mercury as such did not increase the permeability of the balloons. It acted merely

thoroughly with carbon dioxide so that the wall of the balloon was thoroughly saturated with this gas did not prevent the permeation of the gas through the wall of the balloon into its lumen

- 8 *Experiment 8* To determine the effect of hydrogen sulfide upon the balloons of intestinal tubes

Result Latex balloons are highly permeable to hydrogen sulfide. In the presence of mercury a mercuric sulfide forms and the walls of the balloon become discolored and adherent with the result that no cavity remains to hold the gas. Perfusion of the balloons for four days did not change the degree of permeability of the balloon to any appreciable degree. Neoprene G balloons proved to be far less permeable to hydrogen sulfide than latex rubber balloons. When hydrogen sulfide gas was placed within a balloon the speed with which it diffused out was so rapid that this gas becomes of little clinical importance.

- 9 *Experiment 9* To determine whether the bowel prevents the outward diffusion of gas from within the balloon of the intestinal tube. To also determine the effect of submerging the gas inflated balloons in water and the effect of exposing gas filled balloons to air.

Result There was little or no difference in the speed of diffusion of the gases from within the balloons exposed to air or submerged in water. There was no difference in the speed of outward diffusion of the gas from the balloons whether such balloons were within the bowel submerged in water or exposed to air.

Clinical Applications of the Experimental Data

As a result of our experimental studies we can state that all balloons of the long intestinal decompression tube are permeable to intestinal gases in the fashion noted by van Amerongen. The permeability for each specific type of rubber membrane and each specific kind of gas has been determined with such exactness that a formula has

been set up. It being reduced to a formula whose factors are known, the quantity of gas that diffuses through a specific type of rubber balloon can readily be computed. Carbon dioxide and hydrogen sulfide are the gases most likely to diffuse into the balloons of intestinal decompression tubes because of their high degree of diffusibility and because of the markedly higher concentration of these gases within the obstructed bowel as compared to their concentration within the balloon. Nitrogen and oxygen within the bowel do not vary widely from the concentration found within the balloon which contains air. As a result the permeation of the latter gases into the intestinal tube balloons would not be expected to be rapid. The hydrogen sulfide which accumulates within the gastro intestinal tract in a much higher concentration than is found within the balloon of the decompression tube is a gas which diffuses through the rubber membrane with such speed and rapidity that it does not constitute a problem.

Attempts to prevent the intake of carbon dioxide into the balloon by perfusing it with this gas are doomed to failure. The use of Neoprene G rubber in the manufacture of intestinal tube balloons will decrease the effect of this gas since Neoprene G is only 19 per cent as permeable to carbon dioxide as is latex rubber. In the Cantor tube balloon a mixture of Neoprene and latex is used because it has been found that Neoprene loses its elasticity upon stranding and tends to disintegrate very rapidly. By combining the markedly decreased permeability of Neoprene with the elasticity of latex rubber in the Cantor tube balloons we have a balloon which is only a fifth as permeable as the balloons made of latex rubber.

The experimental study showed clearly that if an intestinal decompression tube balloon becomes inflated as a result of carbon dioxide or hydrogen sulfide the passage of a second intestinal tube to decompress the circumjacent bowel should result in the outward diffusion of the hydrogen sulfide or the carbon dioxide from within the balloon into the bowel. The speed of diffusion of the hydrogen sulfide is so fast that it is doubtful whether this gas enters into the problem at all. The only possible influence this gas might have is suggested by

our observations that balloons filled with hydrogen sulfide rapidly lost this gas in less than four hours. If these balloons were then permitted to lie exposed to the air they took up some of the air. Thus diffusion of air into balloons formerly filled with hydrogen sulfide might constitute a problem in intubation or the presence of an air-filled balloon may cause obstruction since 80 per

cent of the air consists of nitrogen gas which is very slowly permeable through rubber. Once nitrogen has accumulated within the balloon of an intestinal tube many days of bowel decompression are required before any appreciable loss of nitrogen occurs from the balloon. This accident can be prevented by using a 21 gauge needle puncture in the wall of the balloon.

GASTRO-INTESTINAL INTUBATION

Intestinal intubation and decompression of the gastro intestinal tract must never be regarded as a substitute for surgery. Although intestinal intubation and decompression may be the sole method of treatment for the non mechanical types of paralytic ileus they are merely an adjunct to the surgical treatment of mechanical intestinal obstruction. The fact that such an obstruction is classified as non-strangulating or strangulating should have no bearing on this rule.

Although the long intestinal decompression tube is of inestimable value when properly used and may even be a life saving instrument the indiscriminate or prolonged use of this tube can have very harmful consequences. The needless waste of time in a futile attempt to pass the decompression tube can prove dangerous for the patient. Experience has amply demonstrated that the surgeon who uses the decompression tube as a substitute for operation assumes a far greater responsibility than the surgeon who insists upon operation in all cases of suspected intestinal obstruction. The middle of the road course to be followed in all these cases is to use the intestinal decompression tube as an adjunct of treatment but not as a method *per se*. In addition it must be noted that attempts to pass an intestinal decompression tube upon a moribund patient are doomed to failure from the beginning. The method of intestinal decompression by means of an intestinal decompression tube should only be used in those patients who are in reasonably good condition and in whom one might expect the gastro intestinal tract to resume its normal function.

The proper use of the intestinal decompression

tube results in internal enterostomy and obviates the necessity for external enterostomy with its associated dangers of local peritonitis and the late stenosis which occasionally occurs. In addition it has been demonstrated that the intestinal decompression tube is the most efficient agent used today for decompressing the gastro intestinal tract in cases of paralytic ileus due to any non mechanical obstruction. In such cases enterostomy is of little value since it merely decompresses one loop of bowel leaving the remainder of the gastro intestinal tract distended. From our experience in the management of all types of intestinal distention we have been led to the conclusion that enterostomy has little or no place in the modern management of intestinal obstruction. For a further discussion of enterostomy the reader is referred to Chapter 20.

In any case where a diagnosis of strangulating obstruction has been made clinically persistent attempts in the passage of the intestinal decompression tube for longer than about six hours are very dangerous. It has been shown experimentally that the bowel will maintain its viability for the first six or seven hours even with complete interference to its blood supply. Although surgery is indicated at the earliest moment for such cases this does not mean immediately upon entrance to the hospital. Generally a period of four to six hours should elapse during which time water and electrolytes should be given intravenously in an attempt at hydration and correction of electrolyte imbalance and blood should be given to replace lost plasma and correct anemia if present. During this same period of time an intestinal decompression tube can be passed and vigorous attempt made to

get it into the gastro intestinal tract beyond the ligament of Treitz in an effort to control distention.

The presence of this tube in the upper bowel not only removes the swallowed air which follows most surgical procedures and causes much of the postoperative distention but the downward passage of the tube proximal to the point of strangulation is of value in keeping the bowel decompressed. Such cases frequently require bowel resection. The intestinal decompression tube within the bowel constitutes an internal enterostomy which not only keeps the bowel proximal to the point of anastomosis decompressed but also protects the suture line by so doing.

In nonstrangulating mechanical obstruction attempts at passage of the long intestinal decompression tube may be safely carried on for 24 hours. If the patient improves during this period of time the long tube will usually be found sufficiently far down the gastro intestinal tract to permit adequate surgery for the mechanical obstruction.

The studies by Wangensteen and Paine using the gastroduodenal tube have emphasized the fact that deflation of the stomach and duodenum often decompresses the proximal jejunum. In addition it has been shown that when the obstruction is due to kinking by adhesions which were perpetuated by the distention itself, gastroduodenal decompression may relieve the obstruction. It was soon realized however that only the upper regions of the gastro intestinal tract could be decompressed by this method. For a large group of patients gastroduodenal suction was not sufficient to relieve the distention. For another group although the distention was relieved the mechanism and the location of the obstructing lesion were unknown. For cases such as these the long intestinal decompression tube was developed in order to provide more adequate and extensive decompression of the gastro intestinal tract. Once the intestinal decompression tube has advanced far down the gastro intestinal tract not only can decompression be obtained but a diagnosis of the obstructing process can also be determined by the injection of small amounts of a radiopaque medium.

USE OF GASTRODUODENAL TUBE

The use of the duodenal tube for diagnostic purposes was an American innovation. Prior to the development of these tubes gastric tubes had been used extensively in Europe and America to remove poisons and other material from the stomach as well as to introduce food into it. In an attempt to intubate the duodenum Turck presented a curious and complicated instrument called a *Gyromele* in 1894. This instrument consisted of a revolving flexible steel cable tipped with a spiral spring and sponge. The cable was encased in a rubber tube and was fitted with a drill arrangement at the proximal end to enable it to be rapidly rotated. Originally designed to outline the stomach by palpation through the abdominal wall this instrument went through many modifications and with it Turck claimed to have intubated the duodenum and obtained duodenal secretions. However few men other than Turck ever used it.

Hemmeter reported experiments that he had carried out in 1896 in an attempt to intubate the duodenum. He first introduced into the stomach a balloon shaped like a stomach with a groove running along its lesser curvature. Through this groove a second rubber tube was passed into the

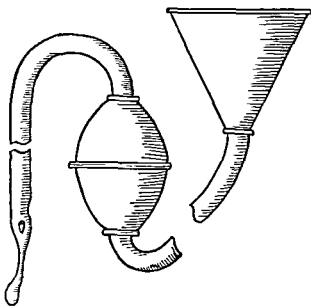


FIG. 273. Gerry stomach pump. Leader at end of tube served function of bolus.

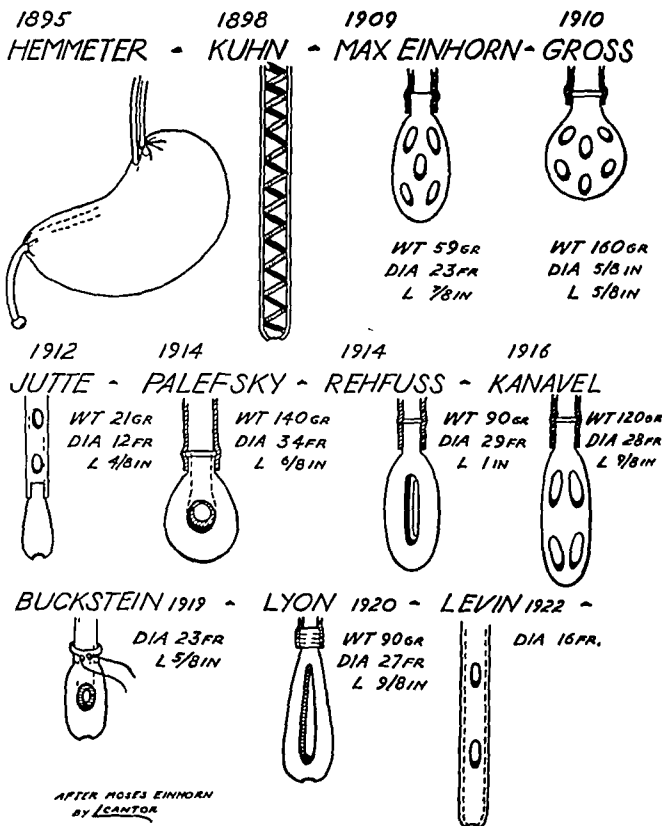


FIG. 274 Various tube heads 1895-1922

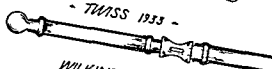
duodenum To prove that the second tube actually entered the duodenum Hemmeter took X rays of the tube *in situ* Experience proved this tube to be cumbersome and impractical In 1898 Kuhn described a rubber covered spiral spring tube and claimed consistent success at direct catheterization of the pylorus The method was not well received by the profession because it was believed to be impractical

The modern duodenal tube was presented almost simultaneously by Gross and Imhorn Imhorn used a long thin rubber tube to which he attached a small metal olive Gross proposed a tube which differed only in having a heavier metal olive The tubes developed by Imhorn and Gross initiated another period of experimentation and study of gastro-duodenal pathology Literally dozens of new varieties of gastroduodenal tubes were developed throughout the world The literature became voluminous after 1910 Everyone who used the duodenal tube believed that he could improve its construction or the technic of its use

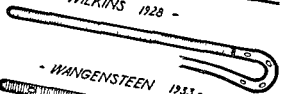
HOLLENDER 1923 - MOSES EINHORN 1926 1927



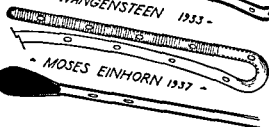
- TWISS 1933 -



WILKINS 1928 -



- WANGENSTEEN 1933 -



- MOSES EINHORN 1937 -

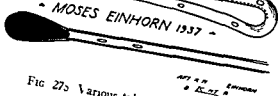


Fig 275 Various tube heads 1923-1937

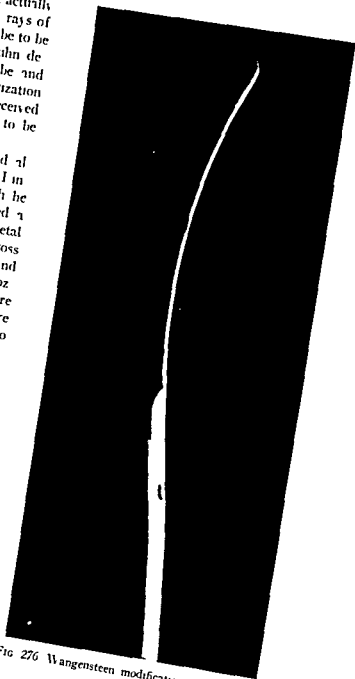


Fig 276 Wangensteen modification of Levin tube

Consequently between 40 to 50 different duodenal tubes were described in the French German English and American literature Most of these were variations of the original tubes and utilized the principle of a weighted tip pulling upon a soft rubber tube The differences in the tubes lay in the changes in shape, composition and weight of the tip The types of tip ranged in composition from ivory to the lead enclosed ball of Moses

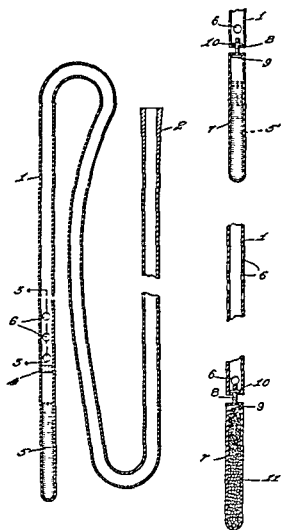


FIG. 277 Wilkins tube

Imhorn with gold silver bead brass and gold plated metal also represented. Weights of the tips ranged from 33 to 170 grams. In 1912 Hess described the use of a small Nelton catheter for duodenal intubation in infants. The anatomic simplicity of infants' stomachs made this process easy to carry out. In 1921 Levin described his smooth catheter tipped duodenal tube for adults. This tube has stood the test of time and has at present replaced all other gastroduodenal tubes because it is simply constructed being made of one piece of rubber and because it can easily be passed through the nose. With the advent of plastics a tube of the same design was proposed by Koslow.

Speed of intubation has been the goal of all who have used duodenal tubes and has been the reason for the multiplicity of types developed. Imhorn

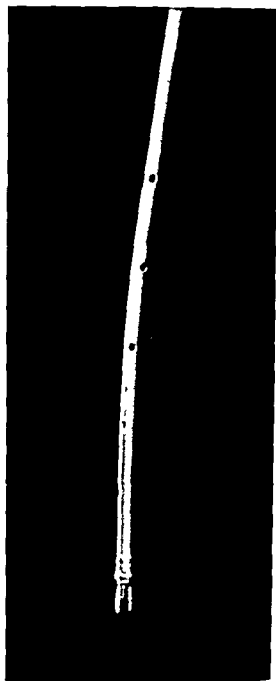


FIG. 278 Jutte tube

passed his tube through the mouth in the evening and allowed 10 to 12 hours for peristalsis to carry it into the duodenum. Lippman claimed to be able to successfully intubate the duodenum in 15 to 30 minutes. Wangenstein used a tube of the Levin design with the tip impregnated with lead to add to its weight. Wilkins proposed a single lumen tube with a mercury filled end and Devine reported the use of a duodenal tube with a tip made

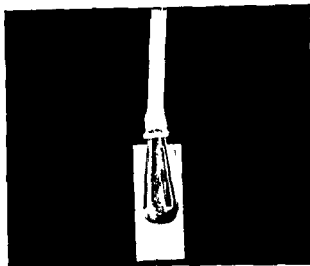


FIG 2/9 Lyons tube

of a highly magnetic permeable metal. This tip was used with an Ahnico Five fixed magnet which was supposed to pull the tip of the tube through the pylorus. A similar tube had been proposed by Mayer 10 years previously.

A comparative study of four different duodenal tubes was carried out by Lake in the course of 390 diagnostic duodenal drainages to determine the differences in the success and speed of intubation. The tubes he used were the Rehlfuss tube, the Swiss tube, the Levin tube, and the Moses Einhorn tube. The weights of the tips of these tubes are: Levin tube—no weighted tip; Swiss tube—69 grams; Rehlfuss tube—74 grams; and the Moses Einhorn tube—150 grams. From his study, Lake concluded that there was no significant difference in the percentage of success with any of these tubes in a 2 hour period and that metal tips of various weights had no advantage over a plain rubber catheter. He found that all the weighted tips, regardless of their specific weights, proceeded to the duodenum with equal rapidity and that nothing was gained by increasing the weight of the tip to 150 grams. However, the plain tipped Levin tube appeared to proceed to the duodenum at a slower pace. Lake therefore concluded that a weighted tip seemed to hasten the entrance of the duodenal tube into the duodenum, although increasing the weight of the tip had no appreciable effect.

Method of Using Tubes

The Levin tube and the plastic variation of this tube, the Koslow tube, are the only gastroduodenal tubes in common use for purposes of decompression. The technique we use in the passage of the Levin tube has been simplified since we do not expect to pass it through the pylorus. The steps in the passage of these tubes are:

1. Inject the patient with $\frac{1}{16}$ grain of morphine sulfate and $\frac{1}{100}$ grain of atropine sulfate. This allays nervousness and offsets sphincteric spasm.
2. Examine the nasal passages and select the side presenting the largest lumen with the least engorgement of turbinates for the passage of the tube.
3. Swab the nasal passage selected with 1 per cent Pontocaine and 2 per cent ephedrine in oil. This shrinks the turbinates and produces sufficient anesthesia to permit the painless passage of the tube.
4. Sit the patient up and hyperextend the neck. Insert the tube well into the nose. When the tip of the tube reaches the pharynx, the patient is permitted to drink water.
5. During the process of deglutition, the tube is slowly but steadily passed downward into the stomach. Suction is now started. Sufficient tubing is passed to permit the tip to reach the pylorus. The passage of too much tubing may result in a kink in the tube with a resultant obstruction or the tip of the tube may be pushed proximally and come to lie just below the cardiac opening. Poor decompression results in either case.

We do not insert iced tubes. A tube at room temperature is much more pliable and hence less uncomfortable for the patient. This is especially true of the plastic tubes. The length of tube required to reach the pylorus may vary from 50 to 107 cm. However, the range of 60 to 75 cm. will give the best results.

Various types of stylets or sounds have been proposed for use with gastroduodenal tubes in an effort to pass them under fluoroscopic control. Among these are the Schlumberger and Camus

ounds who e proponents claim to successfully intubate the duodenum in less than an hour in most cases

Water and Electrolytes During Use of Gastro duodenal Tubes

Suction applied to an indwelling gastroduodenal tube invariably produces a loss of large amounts of water chloride and potassium from the stomach in those cases in which the tube remains within this viscus. In addition patients vomiting may also lose large amounts of bile and pancreatic juice as a result of reflux of these secretions into the stomach. In those cases in which the gastroduodenal tube passes through the stomach and comes to lie in the duodenum which occurs in approximately 60 per cent of the cases with peri-

stalsis this water and electrolyte loss may be considerable. As a result, unless closely supervised these patients develop a severe degree of dehydration and alkalosis due to chloride loss with hypokalaemia. In any patient intubated by gastro duodenal tube it is essential that the sodium chloride potassium and hematocrit levels be determined at frequent intervals so that proper replacement of the water loss and correction of the electrolyte imbalance can be effected.

Patients subjected to continuous gastric suction by means of a gastroduodenal tube should not be given water freely by mouth. Giving water increases the chloride loss in such patients thus increasing the degree of alkalosis. If the mouth is to be moistened cool normal saline solution may be given.

Indications for Use of Gastroduodenal Tubes

The gastroduodenal tube whose tip usually remains within the stomach or duodenum can decompress the bowel when two conditions are present. First it is effective for all obstruction and distention from any cause proximal to the ligament of Treitz. Secondly, it is effective for cases of early obstruction in which the distention is mainly gaseous.

When the gastroduodenal tube is used to decompress the stomach and duodenum in obstruction or ileus proximal to the ligament of Treitz it is not necessary for the tip of the tube to leave the stomach. The reflux of duodenal contents into the stomach permits satisfactory decompression of the distended portion of the gastrointestinal tract under these conditions.

In obstructions distal to the ligament of Treitz the only indication for the use of the gastroduodenal tube is in cases of early postoperative distention or early obstruction. In these cases the distending element is swallowed air. Here the gastroduodenal tube finds its field of greatest usefulness because of the well known tendency of gases to flow from areas of high pressure to those of low pressure until equalization of pressures occur. As a result the gastrointestinal tract would become one continuous gaseous chamber permitting distal gas to pass upward to the point at which the tube had already decompressed the bowel.

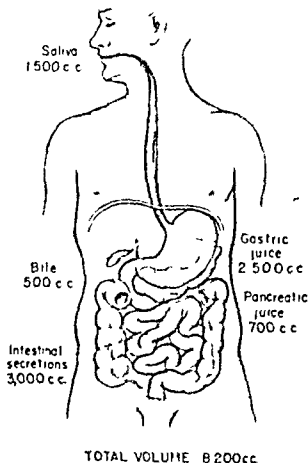


FIG 280 Continuous suction may deplete the patient of many vital fluids and electrolytes. This illustration depicts the tremendous loss which might result if siphonage is continued over a long period.

Paine demonstrated that suction applied to a gastroduodenal tube would not bring the pressure in the terminal ileum to zero in experimental animals although there was a reduction in the intraluminal pressure in the ileum. From this he concluded that although suction when applied and maintained through a gastroduodenal tube (Levin) was highly efficient in decompressing the stomach alone it was necessary to insert the tube through the pylorus into the duodenum to obtain satisfactory decompression of the small bowel. The rhythmic contraction and relaxation of the pylorus are cited to explain the fact that decompression is often obtained when the duodenal tube remains in the stomach. During the period of relaxation gas could be drawn up into the stomach by suction. Paine noted under clinical conditions that often 12 to 36 hours are required to pass the duodenal tube through the pylorus with little or no decompression during this time.

The mechanism believed to be operative in the use of the duodenal tube is described by Paine as follows: *The negative pressure at the end of the tube created by suction causes the bowel about the tube to collapse. Then if peristalsis forced gas up into the collapsed segment of bowel decompression took place. For this reason optimum results are obtainable when peristaltic activity is present and the poorest results are obtained when peristaltic activity is absent as in paralytic ileus.* Paine used a wide variety of negative pressures in his experimental studies and found that the only contra indication to high suction pressures was the collapse of the rubber tubing. In no case was necrosis of the bowel wall as a result of sucking of the mucosa into the openings of the tube noted either in experimental animals or at autopsy. *Our experience has been similar to that of Paine.*

When the duodenal tube was used in the treatment of intestinal distention due to liquid as well as gas it was found that there was a definite decrease in the efficiency of the tube. Since in most surgically treated cases of intestinal stasis the intestinal tract is distended with gas and liquid the mechanism of deflation of the bowel by the tube is different from that when distention is caused by gas alone. With fluid in the bowel the loops of

intestine come to lie in every conceivable plane giving rise to different hydrostatic pressures. The presence of water hose kinks in these loops of bowel tends to create multiple closed loop obstructions instead of one continuous passage. Once formed such kinks are maintained by the weight of overlying loops of bowel and constitute a real obstacle to the equalization of hydrostatic pressure by suction proximal to it. With suction the elasticity of the bowel causes it to contract adjacent to the point of suction as soon as the gas and fluid in the loop are removed. When this occurs decompression ceases until peristalsis or manipulation redistends the collapsed bowel. Thus the duodenal tube can only act by a reflux of the intestinal stream. This is exactly the opposite from the normal we strive for. For this reason in those cases of late intestinal obstruction or paralytic ileus in which there is the greatest need for decompression the gastroduodenal tube fails.

Contra indications to Use of Duodenal Tube

There are many surgeons who believe that no contra indications exist for the use of the gastroduodenal tube in the management of intestinal obstruction. This was true when no better or more efficient instrument was made for use in such cases. However the long intestinal decompression tube now satisfies all the requirements for efficient intestinal decompression in intestinal obstruction. As a result the gastroduodenal tube is contra indicated in all late obstructions distal to the ligament of Treitz. It should never be used as a decompression instrument in obstruction of the colon. It should never be used in the management of paralytic ileus in which the distending elements are liquid and gas.

Complications Due to Use of Gastroduodenal Tube

Although the duodenal tube is a valuable instrument when properly used its use is not without some danger. Complications have decreased since the metal tip is no longer used. A metal tip is traumatic not only to the esophagus but also to the gastro intestinal mucosa when it presses upon it. Nonetheless even the most simple type of duodenal tube the Levin tube or the plastic Koslow

tube is not without danger. The various complications that result from the use of these greatly simplified tubes are not restricted to any one portion of the gastrointestinal tract. In most cases these complications are of minor consequence although in some instances serious results occur. Despite this the good results produced by these tubes make their use mandatory. In many cases complications can be avoided by removing the tube at the earliest possible moment immediately after its use is no longer required. The prolonged and careless use of tubes is responsible for many of the complications.

Knotting of the Tube The Levin and Koslow tubes are likely to undergo knot formation. There have been many reports in the literature describing knot formation with all types of gastroduodenal tubes. Generally such knots are not of serious consequence except that they make removal of the tube difficult and cause a complete loss of function in the tube. Among these reports are those of Billing, Paviot and Levrat, Chaffee, Francesco, Molino and Uribe. In some cases the knot may be of such a size that it becomes difficult or impossible to remove the tube from above. In this event the tube must be cut off at the nose and permitted to pass per rectum. This always occurs within 1 to 15 days except in those cases in which there is a mechanical obstruction distal to the end of the tube. Even a partial obstruction will arrest the excretion of the tube. In cases of this type the coiled tube can sometimes convert the partial obstruction into a complete one. For this reason whenever there is any doubt as to the integrity of the intestinal tract distal to the end of the tube it is best to inject the patient with 100 grains of atropine sulfate and ample sedation and then remove the tube from above. This can usually be done. When the knot reaches

the oropharynx it should be fished out through the mouth and removed. No attempt should be made to pull the knot out through the nasopharynx.

Nose and Nasopharynx The Levin tube and Koslow tube are customarily inserted through the nose down the nasopharynx, oropharynx, esophagus and into the stomach. The presence of a foreign body in the nose and nasopharynx may result in an inflammatory reaction of the nasopharyngeal mucosa in the region of the Eustachian tubes. This is apt to result in the development of sinusitis or otitis media. This complication is particularly likely to occur in infants and children because of the relatively large size of the ostia of these recesses. Because of the reaction of the nasopharyngeal mucosa to the presence of any foreign body it is best not to pass such tubes through the nasopharynx in any individual with a head cold or chronic sinusitis. In such cases oral intubation will avoid the additional inflammatory reaction. Nosebleed of varying degrees may be caused by the passage of duodenal tubes. In infants the bleeding may be troublesome enough to necessitate the removal of the tube. The use of a duodenal tube in smokers is particularly annoying. These people usually present congested nasal mucosa as well as markedly congested pharyngeal mucosa. As a reaction to the duodenal tube there is likely to be a considerable outpouring of mucus in such cases. This is annoying to the patient and may even become dangerous if a mucous plug is inadvertently aspirated into the tracheobronchial tree. Bronchopneumonic and atelectatic results may appear. To prevent some of these complications the tubes should be kept moist. Abscess of the nasal septum and ulceration of the septum have been reported as a result of trauma during the use of duodenal tubes. The healing of such lesions may result in the formation of fibrous adhesions of the septum to the lateral nasal wall.

Esophagus Superficial ulcerations in the esophagus are not uncommon as a result of repeated passages or prolonged use of the duodenal tube. Vinson reported three cases of stricture of the esophagus in patients intubated for relatively long periods of time. Although superficial ulcerations have been observed post mortem along the entire length of the esophagus, generally the distal



FIG. 281. Kehus tube. Note knot formation at the end of tube which completely obstructs it.

esophagus is most commonly involved. For this reason following the removal of the tube cicatrizing stenotic areas may be found along the esophagus. A complaint of difficulty in swallowing after the removal of the tube is a symptom. In these cases esophagoscopy is essential so that such areas of stenosis can be found and treated.

Rupture of esophageal varices may occur during the passage of the duodenal tube. Although it is far more likely to occur when a tube with a metal tip is used it can and does occur during the passage of a Levin tube or a Koslow tube. For this reason it is desirable in intubating patients in whom there is a suspicion of esophageal varices to use extreme gentleness and much water during the passage of the tube. No force should be used. The tube should be permitted to slide down the esophagus with the act of deglutition upon the bolus of water swallowed. In patients of this kind it may be best to avoid the use of any tube unless definite indications requiring intubation are present.

Larynx. The most feared complication of intubation with any tube is laryngeal obstruction. Iglier and Molt reported 10 cases of injury to the larynx as a result of the prolonged presence of the tube. Eight of these cases developed laryngeal stenosis. They noted that the obstruction could be rapid or slow but generally occurred several days after the removal of the tube. In these cases the tubes remained in place from 9 to 20 days. In such cases tracheotomy may be required. Chaffee reviewed the literature and collected 19 cases of laryngeal obstruction caused by the presence of tubes in the esophagus. In one case reported by Holinger and Loeb a tracheotomy was required seven weeks after the removal of the Levin tube because of laryngeal obstruction. In this case the tube had been in the esophagus only four days. Aside from the fact that many of the cases required permanent tracheotomy the 25 per cent mortality rate as associated with this complication gives some idea of its seriousness. Figi reported that of 42 patients with severe stenosis of the larynx and trachea at the Mayo Clinic from 1931 to 1947 3 were due to gastro intestinal intubation. In all of these cases hoarseness, pain upon swallowing, edema of the larynx and respiratory

obstruction developed several days to a few weeks after the removal of the tube and emergency tracheotomy was required.

Morrison described a case of cricoid chondritis when the tube was left in place too long. Wangensteen reported two cases of injury to the arytenoid cartilage due to prolonged intubation. In all cases reported pressure necrosis of the esophagus at the region of the body of the cricoid cartilage was found. Perichondritis and ulceration of the mucous membrane follows and finally subglottic stenosis of the larynx occurs. These patients become hoarse, have difficulty in breathing, develop a croupy cough and complain of difficulty in swallowing. The most dangerous of these complaints is the difficulty in breathing. It is for this that tracheotomy may be urgently needed.

Repeated attempts at forcible insertion of gasroduodenal tubes with blunt and hard tips may result in a perforation of the piriform sinus.

Perforation of the Stomach. Chaffee reported a perforation of the anterior wall of the stomach by a Levin tube. He ascribed the perforation to a pressure necrosis of the stomach wall by the Levin tube. Since the tube had been passed 48 hours prior to surgery it was evident that even this short period of time might be sufficient to produce perforation if the tube caused pressure against a weakened bowel wall. Mahon reported a case in which intubation with a Levin tube for nine days resulted in the development of acute laryngeal obstruction which required tracheotomy two days after the removal of the tube. In addition to the laryngeal obstruction at autopsy this patient was found to have four areas of necrosis on the anterior wall of the stomach just above the greater curvature. Perforation of the stomach had occurred through two of these areas. The cause of death was found to be peritonitis and atelectasis of the lungs. Elason and Welty reported a patient intubated for 36 hours after a small bowel resection. At autopsy a perforation of the anterior wall of the distal esophagus was found. Holinger and Loeb reported a similar case.

Errors and Safeguards in Use of Duodenal Tube

The use of the duodenal tube for the treatment of mechanical obstructions of the terminal ileum

or colon is one of the most serious errors that can be made. The duodenal tube was not designed for this purpose nor does the lower gastro intestinal tract lend itself to decompression by this type of instrument. Thus much valuable time may be lost by starting treatment of these obstructions with a duodenal tube.

Most of the problems associated with the use of duodenal tubes can be avoided by gentleness in the passage and by using a well lubricated tube. Once the tube has been passed it should be kept moist at all times. This is especially important for the nasal portion which should be checked periodically to remove the accumulated mucus.

To avoid pressure necrosis the smallest tube possible should be used. The tube should be soft and resilient. Avoid hardened rubber tubes that have dried out. Only tubing sufficient to bring the tip to the antrum of the stomach should be inserted; too much tubing within the stomach may cause loop formation and the pressure of the hardened tip against the stomach wall may result in perforation.

To avoid many of the nasopharyngeal complications the patient should keep his head flexed forward as much as possible so that the tube will be kept away from the ostia of the Eustachian tubes. This is especially important in children. Remove the tube the moment that its use is no longer required.

USE OF THE LONG INTESTINAL DECOMPRESSION TUBE

The long intestinal decompression tube was devised because of the limitations of the duodenal tube in the treatment of intestinal obstruction. Decompression is far more efficient when the tube is directly at the point of obstruction. The entire length of bowel proximal to the point of obstruction becomes threaded upon the long tube in an accordionlike fashion. As a result the point of obstruction is readily localized not only radiologically but also at the time of surgery. The collapsed bowel on the long tube greatly facilitates operative intervention.

Many types of long intestinal decompression tubes have been developed in the past 15 years in order to intubate and decompress distended pa-

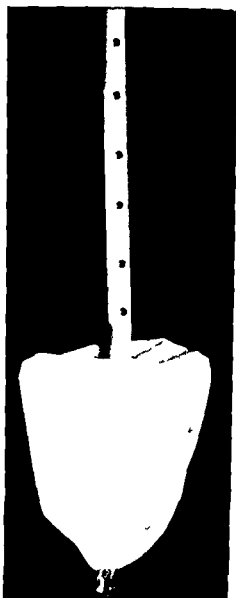


FIG. 282 Miller Abbott tube

tients more successfully and efficiently. Despite the improvement in these tubes, the percentage of successful intubations seems to be in direct proportion to the skill of the person attempting its passage. B. C. Smith, reviewing a large series of cases treated by the Miller Abbott tube, reported a 20 per cent failure rate, whereas Johnston and Miller report very high percentages of successful intubation. In reporting a series of 198 patients with intestinal obstruction treated at the University Hospital at Ann Arbor between the years of 1934 to 1947, Collier and Paxton noted that in only 68 per cent of cases was intestinal intubation successful. It was noted, however, that in the

hands of a skilled and conscientious person with assistance from the radiologist the percentage of successful intubations could be appreciably increased. It should be apparent from this that intestinal intubation is not the simple procedure it is generally believed to be.

History

Intestinal intubation was successfully performed by Schieltema in 1908; the passage of this tube into the duodenum required 15 to 60 hours. Einhorn in 1909 successfully passed an 8 ft. lumen tube into the small bowel to study its contents. This tube was 15 to 20 feet long. In 1926 Van der Reis and Schempera passed a tube through the gastro-intestinal tract and out the anus. Two to six days and repeated feedings were required to accomplish this. They noted that a relatively short length of tube was needed to pass through the entire gastro-intestinal tract.

The balloon-tipped intestinal decompression tube was evolved as a result of the experimental studies of Abbott in 1930 on the effects of drugs on the duodenum. He noted that the distended balloon often passed beyond the duodenum. Miller believed that this spontaneous and rapid passage of a distended balloon might solve the problem of intestinal intubation and suggested that Abbott permit more slack in order that the tube might pass downward more freely. It was soon noted that such tubes passed down into the lower gastro-intestinal tract within a few hours. As a result of these observations Miller and Abbott developed a tube based upon the air-propulsion mechanism which enabled them to study the intestinal contents and carry on physiologic studies with double and triple lumen tubes. They also noted that instead of the 15 to 20 foot length used by Einhorn a tube of half that length was sufficient to reach the cecum. Thus intestinal intubation was established as a practical procedure by 1934.

The surgical possibilities of the Miller-Abbott tube were soon noted. In 1936 Miller, Abbott and Karr described the use of this tube in the management of a patient with bowel obstruction due to carcinoma of the cecum. They noted that the aspiration of gas in front of the balloon initiated peristaltic waves which carried the balloon down-

ward like a bolus. By 1938 articles by Johnston and his co-workers in Detroit, Klein of Mt. Sinai Hospital and Willson of the Mayo Clinic appeared describing the use of the Miller-Abbott tube or modifications of it in cases of intestinal obstruction.

Surgeons everywhere soon realized the advantages of using a long intestinal decompression tube which would pass down to the point of obstruction and the tube began to be used extensively. With the widespread use of the tube it became apparent that although the Miller-Abbott tube was an epoch-making step in the right direction there were some disadvantages to its use. Of these the two most important ones were the difficulty in successfully passing the tube through the pylorus and the ease with which the lumen used for decompression became plugged with intestinal particulate matter because of its small size. The latter could only be prevented by repeated irrigations and constant attention on the part of the nursing staff. An additional disadvantage was the fact that in many cases when the tube had passed far down the gastro-intestinal tract secondary gastric and small bowel dilatation occurred proximal to the end which contained the small decompressing holes. When this occurred the use of a Levin tube to decompress the stomach was required. For these reasons many modifications of the Miller-Abbott

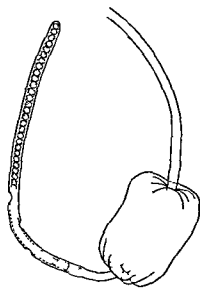


FIG 283 Morton modification of the Miller-Abbott tube. The catheter is weighted with lead shot to facilitate passage of the tube through the pylorus.

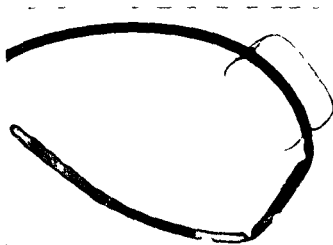


FIG. 284 Radiograph taken of Morton tube. Note the lead shot in the catheter at the end of the tube.

tube and newer techniques for using the tube began to make their appearance.

Types in Use Today

The long intestinal decompression tubes in use today are based upon four different propulsive mechanisms. Each of these tubes was designed and developed to satisfy certain criteria which the surgeon believed necessary to insure intestinal intubation and thus secure adequate decompression. The four types of tubes are represented by the Miller-Abbott tube and its modification, the Johnston tube, the Aguilar tube, the Harris tube, and the Cantor tube.

Miller-Abbott and Johnston Tubes. The Miller-Abbott and Johnston tubes utilized the principle of peristaltic activity as activated by an inflated air-filled balloon. In this way the normal peristaltic activity of the intestinal tract was used to propel the tube downward. The balloon-tipped tube was inflated with air after it had passed through the pylorus. The inflation of the balloon distended the duodenum with the resultant irritation of peristaltic waves which propelled the tube down the bowel like a bolus. Both the Miller-Abbott tube and the Johnston tube were constructed so that a piece of the tube projected beyond the balloon which was affixed to the tube shaft. The tip of the tube was capped by a fenestrated metal head, a small one in the Miller-Abbott tube and a much larger one in the Johnston tube. Explaining the purpose underlying this con-

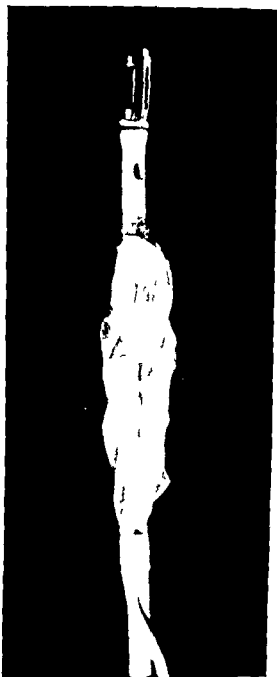


FIG. 285 Johnston tube. Note the projection of part of tube beyond the end of the balloon. Note the large metal piece at the terminal end of the tube.

struction Johnston said: "It is necessary to decompress the gut in front of the balloon in order that the gut may contract upon the inflated balloon which would then propel the tube down the gastro-intestinal tract." The Johnston tube differed from the Miller-Abbott tube in having a larger and heavier metal head and in presenting a small lumen for inflation of the balloon with a larger

lumen used for decompression. In addition unlike the Miller Abbott tube both these lumens were not enclosed in one sheath.

Aguiar Tube The Aguiar tube utilizing the jet propulsion principle as its propulsive mechanism was based upon the concept that if water or any fluid were injected into the end of the tube it would shoot out through the holes at the head end of the tube striking the bowel wall obliquely. The force of this obliquely shooting stream of water or air was used to propel the tube downward. The speed of descent was controlled by increasing the obliquity of the channels through which the water or air emerged, the idea being that with an increase in the obliquity of the emerging stream there would be an increase in the downward propelling force. After the tube head was down the gastro intestinal tract suction would be applied to decompress the patient.

Harris Tube The principle of weight and the effect of gravity upon a weighted tube head was utilized by Harris to devise a single lumen tube. This is a 16 Fr tube with a 6-inch balloon fixed along the shaft at the distal end of the tube. The end of the tube is left open projecting beyond the

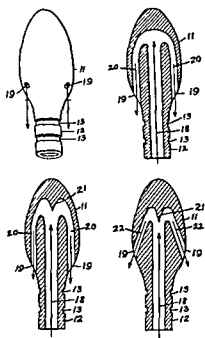


FIG 786 Aguiar tube

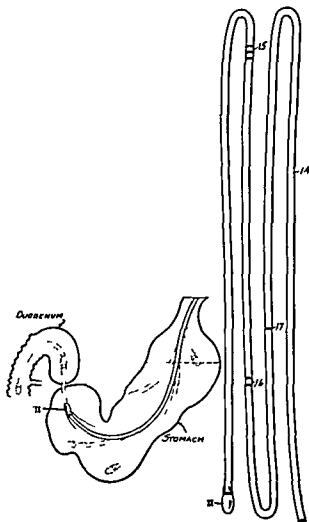


FIG 287 Method of use of Aguiar tube

balloon. Two metal sleeves are inserted within the lumen of the tube at the ends of the balloon.

Cantor Tube The Cantor tube was developed upon the principle of free flow of a mobile cohesive heavy metal confined in a loose balloon tipped tube. It was believed that mercury in itself if given a free range of motion would effectively carry the tube head down the gastro intestinal tract either with or without the aid of peristalsis. For this reason the tube was designed specifically to carry the mercury with successful intubation dependent upon the utilization of all the physical properties of the mercury. This tube was not intended for the study of intestinal physiology but designed solely as an intestinal decompression tube. For this reason a larger luminal diameter



FIG 288 Harris tube. Note the position of balloon along the shaft of the tube with end of tube projecting beyond the balloon.

than ever before was used. In addition an effort was made in designing this tube to meet four criteria for successful decompression. These are:

- 1 A simple single lumen tube without metal parts that could easily be passed down the small bowel.

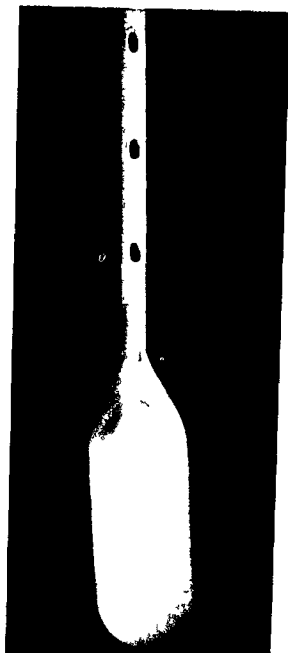


FIG 289 Cantor tube. Note the shape of the holes and the balloon at the distal end of the tube.

- 2 A lumen large enough so that liquid and small particulate matter within the bowel could be suctioned out easily without plugging the tube.
- 3 A sufficient number of holes along the tube so that the bowel could be decompressed along a greater length of tube.
- 4 Holes large enough so that the danger of their becoming plugged by intestinal contents would be reduced to a minimum.



FIG 290 *Top* Cross section of Cantor tube *Below* Cross section of Miller Abbott tube Notice the difference in luminal diameter According to Poiseuille's law the Cantor tube would be four times as efficient a decompressing unit as the Miller Abbott because the flow of fluids through tubes is directly proportional to the square of the diameter

After considerable experimentation it was found that a balloon $2\frac{1}{2}$ inches long and $1\frac{1}{4}$ inches in diameter was the optimum size to hold the mercury. A smaller balloon would not permit a free flow of the mercury and a larger balloon because it could swing around a loop of tube would cause knotting (This would be the result of initially passing too much tubing into the stomach). For successful intubation with this type of tube we must bear in mind the fact that we are utilizing the physical properties of a mobile cohesive heavy metal which runs freely with any movement of the patient and always tends to seek the most dependent level. Successful intubation is assured by maneuvering the patient in such a fashion that the point toward which we want the tube head to go is always downhill. If the patient has good peristalsis rapid and successful intubation always occurs. In the absence of good peristalsis the technique of intubation becomes important if success is to be achieved.

Plastic Tubes In the past eight years as the use of plastics has become increasingly popular three plastic tubes have appeared. These are the Koslow long intestinal tube based upon the same principle of design as the Harris tube, the Honor Smathers tube based upon the same design as the Miller Abbott tube, and the Devine tube which

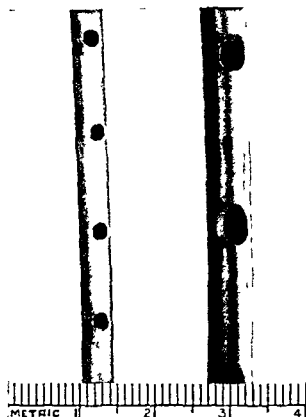


FIG 291 *Left* Miller Abbott tube *Right* Cantor tube Notice the difference between the size of the holes found in the Cantor tube and those in the Miller Abbott tube Notice the elliptical shape of the holes in the Cantor tube The shape and the size of these holes lessen the incidence of plugging from particulate intestinal material

was designed to prevent sucking in of intestinal mucosa during decompression. To prevent this a small plastic inner tube is inserted into the outer perforated tube. Instead of pulling the mucosa into the holes of the perforated tube Devine believes that the suction as it builds up pulls atmospheric air into the tube through the small vent tube.

We do not believe that plastic lends itself to use in intestinal decompression tubes because it lacks both the resiliency and the ability to rebound which is found in fresh rubber tubing. For this reason plastic tends to kink more easily than rubber.

Technics of Intestinal Intubation

Various technics of intubation have been described. In general the technic of intubation varies with the physiologic mechanism upon which the

downward propulsion of the decompression tube is based. The technic of intubation may therefore be described by dividing the tubes into two large groups: (1) the air-filled balloon tube such as the Miller, Abbott and Johnston tubes, and (2) the mercury-bearing tube such as the Cantor tube and the Harris tube. A discussion of the technic of using the Aguilar tube is omitted since this tube is not used in North America because of a reluctance to introduce liquid or air into an intestinal tract already overfilled with air and liquid.

Preparation of the Patient Regardless of the type of tube used, the preparation of the patient is the same. In addition to the preparation discussed in the section under gastroduodenal tubes, there are certain other preparations in the use of the long intestinal decompression tube. An important feature of this preparation consists of explaining to the patient the need for the tube and what we expect to accomplish. This is necessary in order to get complete cooperation. Although

many patients may not be mentally competent to grasp one's explanation yet the frank discussion of our objective with all patients will do much to convert an uncooperative individual into a fully cooperative one. Many of the failures of intubation due to cardio-prism and much of the retching and gagging, generally psychosomatic, can be avoided. In the case of those individuals with whom one cannot discuss the reasons for the use of the tube, one must resort to drugs.

The preparation of the nasal passage is the same as that for the introduction of the gastroduodenal tube.

Great care should be exercised in order to insert and pass the tube with as much gentleness as possible. Considerable difficulty can be avoided if all dental plates are removed before attempting intubation.

Preparation of the Tube Each time a Miller, Abbott or Johnston tube is used, a new balloon should be applied since these balloons lose their

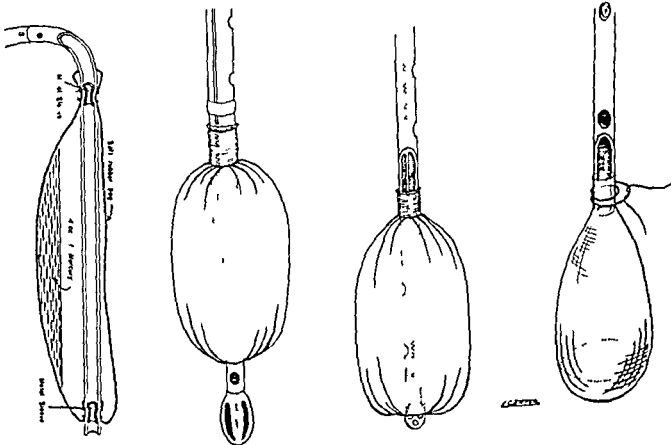


FIG. 292. The tubes are from left to right the Harris tube, Johnston tube, Miller-Abbott tube, and Cantor tube.

efficiency with use as well as becoming discolored and acquiring an unpleasant fecal odor. The method of applying the new balloon is important. The distal end of the balloon is tied on to the decompression tube just behind the distal decompression holes which lie behind the terminal metal tip. The balloon should be straightened out on the tube and when the balloon is tied on proximally sufficient slack must be left between the proximal and distal ties of the balloon to avoid angulation of the tube. A failure to observe this simple precaution results in the inflated balloon producing a kinking of the segment of tubing upon which it is fixed. This angulation or kinking of the tube shaft within the balloon may obstruct the tube distal to this point. Prior to use the balloon should be checked for leaks. Both the balloon and its application to the tube must be airtight. A leak in the balloon or a loose application will not permit proper inflation of the balloon. When this occurs the air propulsion method may fail.

In the preparation of the Harris tube a rubber latex condom 6 inches long is tied on to the distal end of the shaft of the tube leaving its end projecting 2 cm beyond the balloon. A metal sleeve is present within the lumen of the tube at this point. The distal end of the balloon is tied to the tube over this metal sleeve. The balloon is then pulled back over its fixed end and 4 cc of metallic mercury are placed within it. The balloon is then

thoroughly flushed with carbon dioxide, and the balloon is then gently twisted around beginning at the closed end. The carbon dioxide is expelled through the open end by fastening the proximal end of the balloon to the shaft of the tube over a second more proximally placed metal sleeve within the tube. The balloon is securely tied and the tube is prevented from collapsing due to pressure of the ligatures. The tube is now ready for use.

The balloon of the Cantor tube is simply cemented onto the terminal end of the tube. If desired as an additional precaution a silk tie may be applied to the neck of the balloon on the shaft of the tube. Under no circumstances should a tie be placed upon the balloon itself. Since the end of the Cantor tube is completely sealed the mercury must be injected into the balloon with a 21 gauge needle. The following technic is recommended:

- 1 Pour the required amount of mercury into a syringe of adequate size holding the index finger over the tip of the syringe to prevent the loss of mercury.
- 2 Insert the plunger into the syringe.
- 3 Turn the syringe so that its tip points upward and advance the plunger until all the air is forced from the syringe and the mercury fills its tip.
- 4 Attach a 21 or 22 gauge needle to the syringe.
- 5 Pierce the middle of the balloon of the Cantor tube with this 21 or 22 gauge needle.

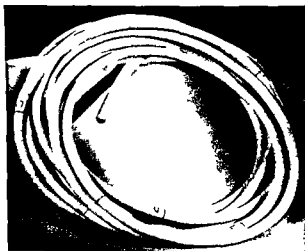


FIG 293 Note end of Cantor tube before assembly. This end is completely sealed off and as a result mercury must be introduced with a syringe and needle.

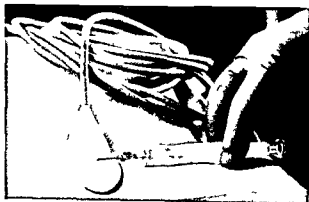


FIG 294 Cantor tube completely assembled. Notice technic of introduction of mercury by the use of a 21 gauge needle attached to a 5 cc syringe. The mercury is injected at the midpoint of the balloon and all the air is then aspirated. The tube is now ready for use.

downward propulsion of the decompression tube is based. The technic of intubation may therefore be described by dividing the tubes into two large groups (1) the air filled balloon tube such as the Miller Abbott and Johnston tubes and (2) the mercury bearing tube such as the Cantor tube and the Harris tube. A discussion of the technic of using the Aguilar tube is omitted since this tube is not used in North America because of a reluctance to introduce liquid or air into an intestinal tract already overfilled with air and liquid.

Preparation of the Patient Regardless of the type of tube used the preparation of the patient is the same. In addition to the preparation discussed in the section under gastroduodenal tubes there are certain other preparations in the use of the long intestinal decompression tube. An important feature of this preparation consists of explaining to the patient the need for the tube and what we expect to accomplish. This is necessary in order to get complete cooperation. Although

many patients may not be mentally competent to grasp one's explanation yet the frank discussion of our objectives with all patients will do much to convert an uncooperative individual into a fully cooperative one. Many of the failures of intubation due to cardio spasm and much of the retching and gagging generally psychosomatic can be avoided. In the case of those individuals with whom one cannot discuss the reasons for the use of the tube one must resort to drugs.

The preparation of the nasal passage is the same as that for the introduction of the gastroduodenal tube.

Great care should be exercised in order to insert and pass the tube with as much gentleness as possible. Considerable difficulty can be avoided if all dental plates are removed before attempting intubation.

Preparation of the Tube I prefer a Miller Abbott or Johnston tube is used a new balloon should be applied since these balloons lose their

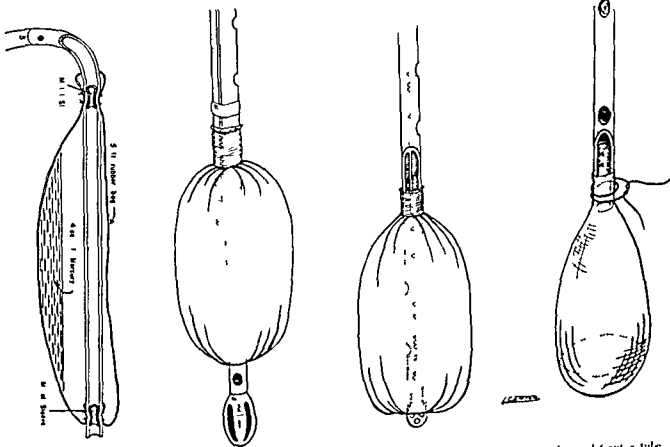


FIG. 292. The tubes are from left to right the Harris tube, Johnston tube, Miller Abbott tube, and Cantor tube.

elasticity with it as well as becoming discolored and acquiring an unpleasant fecal odor. The method of applying the new balloon is important. The distal end of the balloon is tied on to the decompression tube just behind the distal decompression holes which lie behind the terminal metal tip. The balloon should be tightened out on the tube and when the balloon is tied on proximally sufficient slack must be left between the proximal and distal ties of the balloon to avoid angulation of the tube. A failure to observe this simple precaution results in the inflated balloon producing a kinking of the segment of tubing upon which it is fixed. This angulation or kinking of the tube hampers the balloon may obstruct the tube distal to this point. Prior to use the balloon should be checked for leaks. Both the balloon and its application to the tube must be airtight. A leak in the balloon or a loose application will not permit proper inflation of the balloon. When this occurs the air propulsion method may fail.

In the preparation of the Harris tube a rubber latex condom 6 inches long, is tied on to the distal end of the shaft of the tube leaving its end projecting 2 cm. beyond the balloon. A metal sleeve is present within the lumen of the tube at this point. The distal end of the balloon is tied to the tube over this metal sleeve. The balloon is then pulled back over its fixed end and 4 cc. of metallic mercury are placed within it. The balloon is then

thoroughly flushed with carbon dioxide and the balloon is then gently twisted around beginning at the closed end. The carbon dioxide is expelled through the open end. By fastening the proximal end of the balloon to the shaft of the tube over a second more proximally placed metal sleeve within the tube the balloon is securely tied and the tube is prevented from collapsing due to pressure of the ligatures. The tube is now ready for use.

The balloon of the Cantor tube is simply cemented onto the terminal end of the tube. If desired as an additional precaution a silk tie may be applied to the neck of the balloon on the shaft of the tube. Under no circumstances should a tie be placed upon the balloon itself. Since the end of the Cantor tube is completely sealed the mercury must be injected into the balloon with a 21 gauge needle. The following technique is recommended:

1. Pour the required amount of mercury into a syringe of adequate size holding the index finger over the tip of the syringe to prevent the loss of mercury.
2. Insert the plunger into the syringe.
3. Turn the syringe so that its tip points upward and advance the plunger until all the air is forced from the syringe and the mercury fills its tip.
4. Attach a 21 or 22 gauge needle to the syringe.
5. Pierce the middle of the balloon of the Cantor tube with this 21 or 22 gauge needle.

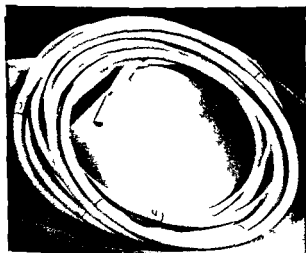


FIG. 293. Note end of Cantor tube before assembly. This end is completely sealed off and as a result mercury must be introduced with a syringe and needle.

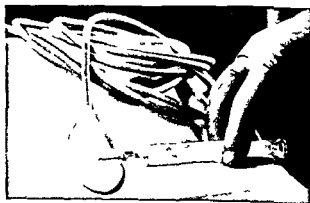


FIG. 294. Cantor tube completely assembled. Note technique of introduction of mercury by the use of a 21 gauge needle attached to a 5 cc. syringe. The mercury is injected at the midpoint of the balloon and all the air is then aspirated. The tube is now ready for use.



FIG 295 Unperforated balloon of intestinal tube immersed in intestinal contents. Note that the balloon is markedly distended with gas.

- 6 Inject the required amount of mercury and then aspirate all the air from the balloon
- 7 Withdraw the needle
- 8 *Do not apply any tie to the balloon itself*

The simplest method for preventing the overdistention of the balloon is to inject the mercury with a 21- or 22 gauge needle. Experimenting with needles of varying size it was found that only a 21- or 22 gauge needle would provide the ideal puncture hole. Holes made by smaller caliber needles would be too small to act as a safety vent if gas were taken up by the balloon whereas larger sized needles made a hole so large that leakage of mercury would occur. With a 21- or 22 gauge needle the mercury does not escape unless the balloon at the site of the hole is stretched during insertion. Even if the balloon becomes distended the vent created rapidly permits the gas

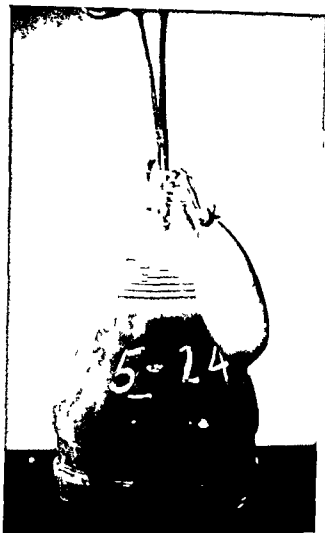


FIG 296 The same balloon as in Figure 295. This balloon was not perforated before being immersed in intestinal contents. Note the marked distention from intestinal gas.

to escape. The site of choice for the puncture was found to be the middle of the balloon equidistant from neck to base. This is the point at which the greatest stretching would occur when the balloon became overdistended with gas. It is impossible to overdistend balloons punctured at this point.

The amount of mercury which should be placed in the balloon of this type of tube varies with the type of patient being intubated as well as with the pathologic process for which the tube is being passed. We have found that it is possible to predict with a fair degree of accuracy the amount of mercury which will result in successful intubation in most types of patients. The amount of mercury



Fig. 297 Note the relative absence of gas in a balloon which had previously been perforated by a 21 gauge needle before being immersed in intestinal contents. A comparison of this figure with Figures 295 and 296 indicates the prophylactic effect of perforating the balloon of an intestinal tube prior to usage



Fig. 298 The use of 5 cc. of mercury in a Cantor tube successfully passed in the management of a patient with normal peristaltic activity

used may vary from 2 to 10 cc depending upon the specific patient. With experience it is possible to classify all patients into one of four groups.

1 In this group are those patients in whom there has been no loss of tone in the gastro intestinal tract nor any impairment of peristaltic activity due to muscular weakness.

2 This group includes all patients who are nervous and high strung.

3 This group is composed of those patients presenting atony or paralytic ileus or loss of the coordinated intestinal movement necessary to propel an intestinal tube.

4 The patients in this group have a condition in addition to intestinal atony which precludes motion and ambulation.

In the first group are found those patients with

mechanical obstruction of the bowel regardless of cause who are seen early in the course of the disease before paralytic ileus set in. Peristaltic activity is active in these patients. For individuals in this group 4 to 5 cc of metallic mercury within the balloon of the tube are sufficient to carry it into the jejunum in 24 hours or less. In many of these patients the head of the tube may be found in the ileum at the end of 24 hours. Patients in this group are usually easily intubated and the percentage of failures is small. Increasing the amount of mercury used to 8 or 10 cc in patients in this group increases neither the speed with which the tube passes nor the distance it moves along the intestinal tract in a 24 hour period.

The second clinically recognizable group is composed of all those patients who are high strung and

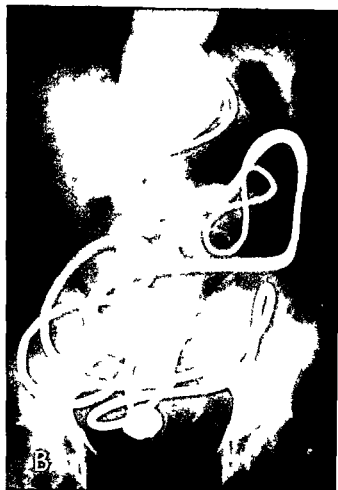


FIG. 299 A second tube was passed containing 8 cc of mercury. Note that the second tube is in relatively the same position as the first (see Fig. 298) indicating that in this type of case increasing the amount of mercury is of no value.

nervous. For this group as for the first, the reason for intubation was found to make little difference as far as determining how much mercury to use in the tube head. Many of the patients develop cardiospasm or pylorospasm so that the tube becomes trapped either in the lower esophagus or the stomach. As long as the sphincters remain in spasm, the tube will not pass if only 4 to 5 cc of mercury are used in the balloon. The patients are apprehensive and greatly fear intubation. In intubating patients in this group we have found it necessary to use from 8 to 10 cc of mercury within the balloon. This amount of mercury will generally bypass any spastic sphincter if the tube head is permitted to press upon it. In a case of cardiac sphincteric spasm, ambulating the patient or standing him up results in a weight of a fourth



FIG. 300 Note the effect of leakage of the mercury. The downward propulsion of the tube is greatly hindered and as a result looping of the tube in the duodenum occurs.

of a pound (9 cc) of cohesive heavy metal (mercury) pushing upon the cardiac sphincter. The fluidity of the mercury and its cohesive power are such that successful passage through the cardiac sphincter is generally obtained in this fashion. If the pyloric sphincter is in spasm, then the position in which the patient is placed depends upon the type of stomach being intubated. In the case of a steer horn stomach with a low lying pylorus, ambulation and motion of the patient result in the tube head pressing upon the most dependent portion of the stomach, which in this position would be the pylorus. If the stomach is of the J type, it is important that the patient be turned on the right side and inclined obliquely, feet downward, with the foot of the bed elevated from 12 to 15 inches. In this position, the first portion of the duodenum runs downhill and the pylorus becomes the lowest point in the stomach. From our observations, it would seem that in intubating patients who are nervous and apprehensive and in whom sphincteric spasm might be anticipated, the per-



FIG. 301 Five cubic centimeters of mercury within the balloon of a tube in a patient (Carl Johnson). Note the arrest of tube in the lower esophagus.



FIG. 302 Plastic tube containing 5 cc of mercury. Note the tube held at the pylorus but a successful passage

centage of failures will be materially reduced by the use of 8 to 10 cc of mercury within the balloon of the tube.

The third group of patients all present one thing in common—an atony of the gastrointestinal musculature or loss of the coordinated intestinal movement required to propel an intestinal tube downward. This condition is clinically classified as either atonic ileus or paralytic ileus. These patients are described as having a silent abdomen because no peristaltic sounds are audible. In intubating patients in this group we have found it necessary to increase the amount of mercury in the tube head to 8 or 10 cc. In addition these patients must be so maneuvered that the head of the heavily weighted tube head is to go will always be downhill. When this amount of mercury is used in an atonic or paralytic gastrointestinal tract it is essential to ambulate the patient once the tube head has passed through the third portion

of the duodenum. In such patients little help is given by peristaltic activity and dependence must be placed upon the effect of motion and the position of the patient so that the weighted tube head which always seeks the most dependent level will always go where we want it to. Although with the use of 4 to 5 cc of mercury many tubes can be successfully passed in patients in this group the percentage of failures will be high. Since it is so important it becomes highly desirable to pass a tube as rapidly as possible. This can best be accomplished by the use of 8 to 10 cc of mercury in the tube head.

The fourth group includes all those patients presenting some unusual condition. In addition to the atony of the bowel these patients also have an illness which precludes ambulation and motion. Patients with fractures of the vertebra or with malignant growths of the vertebral bodies and involvement of the cord are found in this group. The



FIG 303 The plastic tube (see Fig 302) is removed and the Cantor tube passed with 9 cc of mercury. Note the position of the tube 48 hours after intubation with this second tube



FIG 304 Position of tube head containing 3 cc of mercury. Intubation for paralytic ileus resulting from a malignant invasion of the vertebral body such that motion of the patient could not be obtained

abdomen may be greatly distended. Often sufficient tonus and peristaltic activity may be present to propel a lightly weighted tube downward very slowly whereas a heavily weighted one would remain in the stomach. Since the motion of the patient and hence the effect of motion and gravity on the tube head are not obtainable in this type of patient it is essential to use the least amount of mercury possible. We have found that the use of 2 to 3 cc of mercury in the balloon in these cases usually brings about successful intubation. Suction must be started as soon as the tube enters the stomach so that the stomach is emptied and a sufficient return of tonus is obtained for the passage of a tube so lightly weighted. Although the downward progress of the tube may be slow, excellent decompression will be obtained.

Passage of the Air-filled Balloon Tubes (Miller Abbott, Johnston, Honor Smathers Tubes) The tip of the well lubricated tube is in-

serted into the nasal passage and sufficient tubing is passed until the tube is felt in the nasopharynx of the patient. At this point the patient sits up and takes a drink of water. The swallowing of the water facilitates the downward passage of the tube into the stomach. Generally a few swallows of water accomplish this. It is a good idea to measure the tube against the patient prior to its insertion to get a rough idea of the amount of tubing required for the tip to reach the pylorus. It has been shown repeatedly that this length may vary from 50 to 107 cm, with an optimum range of 60 to 75 cm. The aspiration of gastric contents assures the intubator that the tube is indeed in the stomach. The tube should then be passed until it tips a short distance from the pylorus.

If the nasal passage of the patient is too small to accommodate the metal tip of the John-ton tube a retrograde method of passage is indicated. There are two possible methods. First the metal tip of

the Johnston tube may be removed and the well lubricated balloon portion passed through the nose. This is then pulled out through the mouth, the metal tip reattached and the tube swallowed. Second, the metal tipped tube may be passed through the mouth of the patient and the tube swallowed. A small catheter is then passed through the nose and brought out through the mouth. The proximal end of the Johnston tube with all metal parts removed may then be tied to the catheter which is pulled back through the nose, bringing the proximal end of the tube with it. The metal adaptors are then reattached and the suction may now be applied. Gagging during these maneuvers may be avoided by painting the throat with Pontocaine and in addition by keeping the head bent forward to keep the tube away from the pharynx.

With the tube head in the stomach, the gastric contents are aspirated. Permitting the patient to drink during the process of intubation into the stomach also washes out this viscus, removing gastric particulate material. From this point on, the most difficult phase of the intubation process begins. This is the successful passage of the tube through the pylorus and into the duodenum. Several methods have been proposed to accomplish this.

The first method consists of applying suction to the tube to completely empty the stomach. The tube is then withdrawn until its tip lies well within the stomach cavity. If fluoroscopy is not available

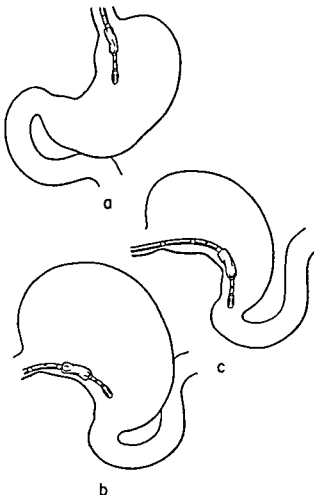


FIG. 306. Technic to facilitate passage of tube to pylorus. (a) with tube tip in this position the stomach is emptied and then inflated with air to produce distention. (b) with patient lying on the right side tip drops through distended stomach. (c) tube is advanced then emptied.

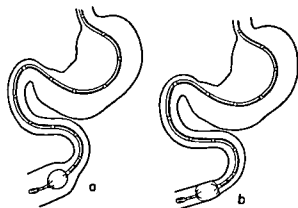


FIG. 305. The tube will not progress through the small intestine if the bowel is not decompressed (a) as it is necessary for gut wall to approximate balloon (b) to propel tube.

to check the position of the tube. Abbott has suggested inflating the balloon with air and withdrawing it until the air-filled balloon meets with resistance at the cardiac sphincter. This is ample proof that the tip of the tube is in the cardiac portion of the stomach. The balloon is then emptied. The patient is placed on his right side and the tube advanced slowly for a distance of 6 inches. The tube end should then be at the pylorus. This position may be checked fluoroscopically in order to be certain that the tube is lying straight and has not coiled within the stomach. If the tube is found to be curled or twisted upon itself, it must be



FIG. 303 The plastic tube (see Fig. 302) is removed and the Cantor tube passed with 9 cc. of mercury. Note the position of the tube 48 hours after intubation with this second tube.



FIG. 304 Position of tube head containing 3 cc. of mercury. Intubation for paralytic ileus resulting from a malignant invasion of the vertebral body such that motion of the patient could not be obtained.

abdomen may be greatly distended. Often sufficient tonus and peristaltic activity may be present to propel a lightly weighted tube downward very slowly whereas a heavily weighted one would remain in the stomach. Since the motion of the patient and hence the effect of motion and gravity on the tube head are not obtainable in this type of patient it is essential to use the least amount of mercury possible. We have found that the use of 2 to 3 cc. of mercury in the balloon in these cases usually brings about successful intubation. Suction must be started as soon as the tube enters the stomach so that the stomach is emptied and a sufficient return of tonus is obtained for the passage of a tube so lightly weighted. Although the downward progress of the tube may be slow, excellent decompression will be obtained.

Passage of the Air-filled Balloon Tubes (Miller Abbott, Johnston, Honor Smathers Tubes) The tip of the well lubricated tube is in-

serted into the nasal passage and sufficient tubing is passed until the tube is felt in the nasopharynx of the patient. At this point the patient sits up and takes a drink of water. The swallowing of the water facilitates the downward passage of the tube into the stomach. Generally a few swallows of water accomplish this. It is a good idea to measure the tube against the patient prior to its insertion to get a rough idea of the amount of tubing required for the tip to reach the pylorus. It has been shown repeatedly that this length may vary from 50 to 107 cm. with an optimum range of 60 to 75 cm. The aspiration of gastric contents assures the intubator that the tube is indeed in the stomach. The tube should then be passed until its tip is a short distance from the pylorus.

If the nasal passage of the patient is too small to accommodate the metal tip of the Johnston tube a retrograde method of passage is indicated. There are two possible methods. First the metal tip of

the Johnston tube may be removed and the well lubricated balloon portion passed through the nose. This is then pulled out through the mouth, the metal tip reattached and the tube swallowed. Second, the metal tipped tube may be passed through the mouth of the patient and the tube swallowed. A small catheter is then passed through the nose and brought out through the mouth. The proximal end of the Johnston tube with all metal parts removed may then be tied to the catheter which is pulled back through the nose, bringing the proximal end of the tube with it. The metal adaptors are then reattached and the suction may now be applied. Gagging during these maneuvers may be avoided by prying the throat with Pontocaine and in addition by keeping the head bent forward to keep the tube away from the pharynx.

With the tube head in the stomach, the gastric contents are aspirated. Permitting the patient to drink during the process of intubation into the stomach also washes out this viscous removing gastric particulate material. From this point on the most difficult phase of the intubation process begins. This is the successful passage of the tube through the pylorus and into the duodenum. Several methods have been proposed to accomplish this.

The first method consists of applying suction to the tube to completely empty the stomach. The tube is then withdrawn until its tip lies well within the stomach cavity. If fluoroscopy is not available

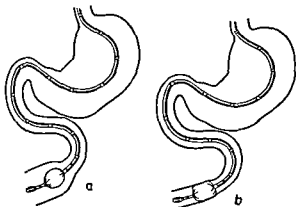


FIG 30a The tube will not progress through the small intestine if the bowel is not decompressed (a) as it is necessary for gut wall to approximate balloon (b) to propel tube

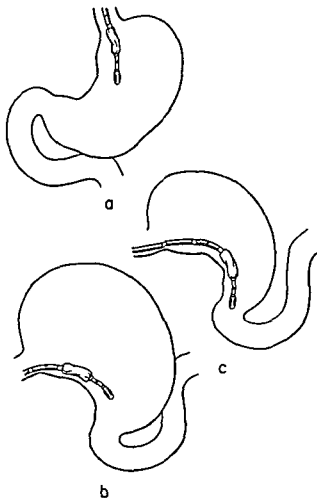


FIG 30b Technique to facilitate passage of tube to pylorus (a) with tube tip in this position, the stomach is emptied and then inflated with air to produce distention (b) with patient lying on the right side tip drops through distended stomach (c) tube is advanced for several inches to pylorus and air is then aspirated

to check the position of the tube. Abbott has suggested inflating the balloon with air and withdrawing it until the air filled balloon meets with resistance at the cardiac sphincter. This is ample proof that the tip of the tube is in the cardiac portion of the stomach. The balloon is then emptied. The patient is placed on his right side and the tube advanced slowly for a distance of 6 inches. The tube end should then be at the pylorus. This position may be checked fluoroscopically in order to be certain that the tube is lying straight and has not coiled within the stomach. If the tube is found to be curled or twisted upon itself it must be

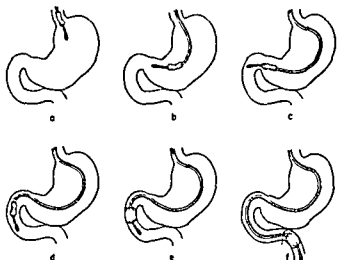


FIG. 307 Normal progress of tube when difficulties are not encountered. (a) Tube tip well through the esophagus. (b) passage through the stomach requires slow advance. (c) entrance into the duodenum by very slowly advancing tube. (d) balloon well into the duodenum before any air is introduced into the balloon. (e) then only a small amount is introduced—approximately 10 cc. (f) when the balloon has passed the duodenojejunal fold the balloon is fully distended (30-50 cc of air).

withdrawn until the balloon is again at the cardia of the stomach. It should be passed 2 cm from the point as before. If the tip of the tube is found to be at the pylorus Johnston reports that it is often possible to pass it directly into the duodenum. To accomplish this the patient is placed in the supine position and the tube is advanced until it appears to turn in the stomach. When this is noted fluoroscopically gently insert and withdraw 1 inch of tube until the pylorus is successfully intubated. Usually this maneuver is successful in passing the tube into the duodenum. Then the tube must be inserted slowly allowing a few minutes to elapse between the introduction of each inch of tube. This is said to advance the tube successfully into the third portion of the duodenum. With the tube in the duodenum the balloon is inflated with 10 cc of air. When the balloon has passed the duodenojejunal flexure the balloon is further inflated with 20 cc of air. If the tube has not passed through the pylorus at the first attempt the patient is returned to bed and turned on the right side. He is permitted to remain in this position for an hour with constant suction applied to the tube. At the end of the hour the balloon is inflated with 3 cc

of air. If it fills easily and without tension it would suggest that the tube is still within the stomach. If however the balloon fills under tension so that the plunger of the syringe moves with difficulty and is readily pushed out it indicates that the balloon is within the duodenum. This may also be shown graphically by attaching a water manometer to the proximal end of the inflated balloon. An increase in pressure due to the relatively small size of the duodenum as compared with the stomach indicates that the tube is in the duodenum. The aspiration of bile from the tube also suggests that the balloon is in the duodenum. However green bile may be aspirated by a tube in the stomach. This is caused by vomiting with reflux of duodenal contents into the stomach. Golden bile on the other hand usually indicates that the end of the tube is in the duodenum and that the bile is obtained directly from the common duct.

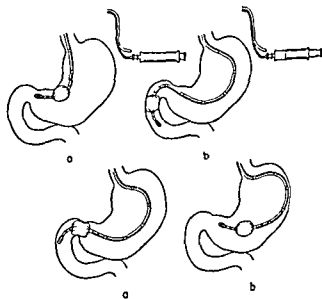


FIG. 308 Tip. With balloon well into duodenum, golden yellow bile may be aspirated from the tube. In the absence of characteristic type of drainage, air carefully injected into the balloon is of assistance in determining position. (a) With balloon in the stomach, fully injected into the balloon is of assistance in determining position. (b) When balloon is in the duodenum a few centimeters of air injected into the balloon will cause the bile to enter the tube and tend to push the plunger out of the syringe if free to move. Full inflation of the balloon before it is well into the duodenum (1) causing tube to pull back into stomach (2).

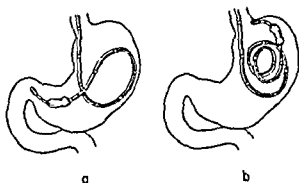


FIG. 309 Results of hurrying tube through the stomach (a) Crossed loop in the stomach causes difficulty in passing tube into the duodenum but does not absolutely prevent it (b) coiling of tube in stomach makes passage into the duodenum almost impossible

The second method consists of having the patient lie on his right side so that the tip of the tube may be directed to the antrum. After four hours 1 inch of tube is introduced through the nose every half hour until a foot of tube has been passed into the stomach. This permits the tip to approach the pylorus slowly. It also allows the tube to be carried into the stomach without coiling. After 1 foot of tube has been slowly introduced an X-ray or fluoroscopic examination is desirable to determine whether the tip has really entered the duodenum. This is the only certain method for knowing

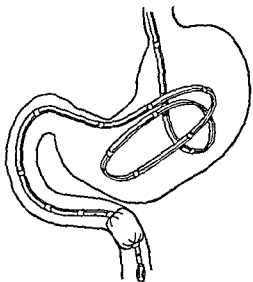


FIG. 310 Coil of tube in the stomach will retard progress of tube in the intestine even if decompression ahead of balloon is adequate and peristalsis is active

whether intubation of the duodenum has been successful.

When the standardized technique described here has been successful the proponents of the air-filled balloon tubes say that the tube may be expected to proceed downward at a speed of a foot an hour. Blodgett reported the passage of the tube to the cecum in 18 hours but noted that in peritonitis with paralytic ileus it may be a matter of days before the air-filled balloon propelled tube reaches the ileum. We have found however that the speed of descent of this type of intestinal decompression tube varies within wide limits depending upon the type of patient being intubated, the pathologic condition present requiring intubation, the tone of the intestinal musculature and the presence or absence of peristaltic activity. In our experience we have found that in intubating mechanically obstructed individuals with vigorous peristalsis or hyperperistalsis the rate of descent will be rapid so much so in fact that within 12 hours the tube head may be found well down in the ileum. In some cases the tube head may be found in the lower ileum within this same period of time. In those cases which are being intubated for paralytic ileus however the descent of the decompression tube is slow. In fact it is so slow that usually the tube will not pass much beyond the upper jejunum if that far in the first 24 hour period. Normally once the intestinal distention has been decreased and normal peristalsis reestablished the further descent of the tube is more rapid until the point of obstruction is reached.

When the standardized technique for passing these air-filled balloon tubes is unsuccessful the intubator may use some of the other methods advocated to assure the passage of the tube through the pylorus. The most commonly employed of these methods are

1. Morgenstern's method. This method consists of sitting the patient up behind a fluoroscopic screen. With his left hand the intubator then pushes up on the greater curvature of the stomach in an effort to bring it to the same level as the pylorus while with his right hand he passes or threads the tube head through the pylorus.
2. Another method consists of inflating the

stomach with air and turning the patient on his right side. Distention of the stomach now permits the tube head whose balloon is air filled to float to the pylorus. Johnston believes that the larger heavier tip in his tube makes this maneuver more successful.

- 3 Abbott suggests that in some cases the stomach can be distended with water. The patient is then turned on his left side, the balloon inflated with air, and the air filled balloon floated to the pylorus at the top of the water. After the tip of the tube has passed through the pylorus, the water is aspirated and the balloon deflated.

- 4 Abbott suggests another method which is the use of a wire stylet inserted into the lumen of the tube to pass it under fluoroscopic control through the pylorus after which the stylet is removed. In using the stylet, the wall of the aspirating lumen of the tube is pierced at the 50 cm mark with a length of 0.4 mm diameter, stainless steel vom Hofe leader wire. The tip of the wire is advanced to a point an inch above the most proximal aspirating hole in the tube. Bend a loop in the other end of the wire so that the tip cannot be advanced any further distally. Thus the terminal 15 cm of Miller-Abbott tube contains no wire. The tube is then passed in the usual fashion until the balloon is well within the stomach. With a temporary adhesive tape patch over the point at which the stylet pierces the tube wall, inject 300 cc of air into the stomach and advance the tube until it lies along the greater curvature. Hold the tube at the patient's nose to prevent it from slipping out. Apply suction now to empty the stomach of air. The stomach then contracts, squeezing the tip of the tube ahead from its position on the greater curvature to its position along the lesser curvature. The tip of the stylet should now be near the pylorus. Then gently draw back the tip of the stylet and by gentle steady pressure on the tube at the nose advance the tip through the pylorus under fluoroscopic control. Withdraw the stylet, patch the hole through which it was intro-

duced with thin rubber cement and inflate the balloon with 10 cc of air. Grafton Smith has recently devised a tube whose tip is controllable by means of a stylet utilizing a hand control to direct it. With this device he has been able to successfully intubate the duodenum in less than 30 minutes.

- 5 Mayer's method. A magnetized head made of Alnico is used at the tip of the tube. The head being magnetized is then pulled through the pylorus by a magnet at the right flank under fluoroscopic control.
- 6 Willson's method. This method uses a heavier tube of larger caliber than that of the Miller-Abbott tube. This tube is introduced with the patient either on the right side or back in the semi-Fowler's position. After the tube head has left the stomach, the balloon is filled with 30 cc of fluid instead of air.
- 7 Morton's method. This proposes the addition of a weighted tip to the Miller-Abbott tube for more successful intubation. His method consists of weighting the tip of a Levin tube for the distal 5 inches with lead shot and then connecting this weighted tip to the end of the Miller-Abbott tube by means of a short metal tube of proper caliber.
- 8 Svendsen's method. Mercury is used in the balloon of the Miller-Abbott tube to weight its head. Four to five cubic centimeters of mercury are placed within the balloon of the air-filled balloon tubes to weight the head.
- 9 Martinetto's method. Martinetto suggests splanchnic nerve block as an adjunct in intestinal intubation. This method is based upon Martinetto's concept that in mechanical obstruction the autonomic system is also involved, accentuating the obstruction by spasm giving rise to zones of atony in other parts or causing intestinal hypersecretion. As a result of the sympathetic nerve (splanchnic) interruption the following results are obtained: (1) stimulation of the tonus and contraction of the muscular walls of the esophagus, stomach and intestine and (2) persistent relaxation of the cardiac and pyloric sphincters, particularly the

pylorus Martinetto uses 1 per cent novocaine to produce the block of the splanchnic nerves with the addition of 3 mg. of atropine chloride. This block is repeated twice a day. Martinetto reported that this method of speeding intubation produced excellent results in 30 patients.

Passage of the Mercury weighted Balloon Tube (Cantor Harris Koslow Tubes) All the mercury bearing intestinal decompression tubes are single lumen tubes which differ in the point of attachment of the balloon. Being single lumen tubes they are four times more efficient as decompression instruments than are the double lumen tubes of the same external caliber. This is in accord with Poiseuille's law which states that the flow of fluids through tubes is directly proportional to the square of the diameter.

The technic of passing these tubes is as follows:

1. Have the patient lie flat on his back with his head hyperextended or sit him up with the neck hyperextended. Remember that the sac with the mercury must run downhill. Feed the sac with the mercury into the external nares. Three techniques may be used to do this:
 - (a.) Grasp the balloon at the tip to allow the mercury to run toward the tie. Now fold the bag to form a spur. Insert the spur far into the nose. Release

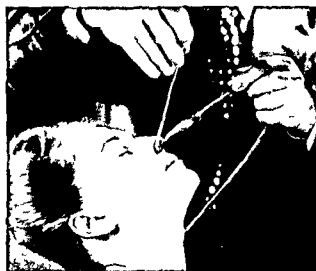


FIG. 312 The use of a cotton tipped applicator in the insertion of the Cantor tube.

the fingers holding the mercury at the back end of the balloon and gently squeeze the balloon into the nasal passage.

- (b.) Push the balloon containing the mercury back into the nasopharynx using a cotton tipped applicator. In doing this be careful that the side of the balloon perforated in inserting the mercury is not stretched. This can be avoided by placing this portion of the balloon inferiorly in the nose.



FIG. 311 Technic of inserting the Cantor tube.



FIG. 313 The use of a bayonet forceps in the passage of a Cantor tube. This is particularly useful in those cases in which there is a perforated septum.



FIG. 314 Note the adaptation of the balloon of the Cantor tube to the maxillary fossa



FIG. 315 Note the presence of the Cantor tube balloon in the nasopharynx

(c) If the patient has a perforated septum the balloon is best inserted into the nasopharynx by a bayonet forceps. When the balloon containing the mercury reaches the nasopharynx the intubator will feel a tug on the tube. At this point sit him up and give him a drink of water. When the patient swallows the water the mercury tipped tube will drop down the esophagus into the stomach. Pass in sufficient tubing to bring the letter S to the external nares. This indicates that the tube is in the stomach. Now connect up the suction.

2 Do not push the tube down. To pass the tube have the patient drink water. When the patient swallows water hold the tube up in the palm of the hand and gently permit it to be carried downward by the process of deglutition. Do not advance it faster than 4 inches every two hours. Too rapid passage



FIG. 316 Notice the downward drag of the balloon of the Cantor tube in the oropharynx



FIG 317 The tube has proceeded rapidly down the esophagus



FIG 318 Notice the effect of turning the patient on the right side and raising the foot of the bed. The tube head has now come to lie at the pylorus

of the tube causes coiling in the stomach and predisposes to knot formation

- 3 With the S at the nose and the tube in the stomach have the patient lie on his right side (without a pillow) and elevate the foot of the bed 12 inches. During this time pass in sufficient tubing to bring the letter P to the external nares. This will bring the bag to the pylorus. Permit the patient to remain in this position for two hours.
- 4 After two hours turn the patient on his back, lower the foot of the bed and place the patient on a back rest. At this time pass in sufficient tubing to bring the letter D to the external nares. This should bring the tube into and down the duodenum. Permit the patient to remain in this position for two hours.
- 5 Now with the patient flat in bed turn him on his left side. Permit him to remain in this position for two hours. Pass 4 inches of tube after the patient has turned on his left side. After this period permit the patient

to move about freely. Motion of the patient greatly aids in the downward passage of the tube.

- 6 Check up X ray to determine the position of the tube six hours after insertion.
- 7 *Do not fasten the tube to the face of the patient.* The tube must be permitted to lie free at all times and the 4 inch portion just outside of the nose must be kept moist with mineral oil.

The maneuvers described above utilize the downhill concept to pass a weighted mercury tipped tube in a J type of stomach in which the first portion of the duodenum runs uphill, the second limb downhill, and the third portion runs from right to left and uphill. Under these anatomic conditions the described maneuvers will constantly keep the balloon containing the mercury running in a downhill course. However in a steer horn stomach the anatomic conditions are changed and the first limb of the duodenum lies in an al



FIG. 319 Following the passage of the tube head through the pylorus and sitting the patient up result in the downward passage of the tube into the second limb of the duodenum. Notice the direction of the second limb of the duodenum.

most direct downhill course from the stomach. In intubating a stomach of this type ambulation alone is sufficient to pass the tube rapidly through the pylorus without the necessity of any of the maneuvers described. An atonic stomach would require the described maneuver if the pylorus were fixed at a high point. However, since the intubator usually does not know the type of stomach being intubated it is a good idea to maneuver all patients as described. Then if a check film shows an unsuccessful intubation one might suspect that a

J type of stomach was not present and ambulation could then be used. If the stomach being intubated is a postgastrectomy stomach ambulation is sufficient to pass the tube head down the distal loop because the outlet of the stomach is at the inferior portion of the vertically lying gastric



FIG. 320 The tube head is now at the duodenojejunal flexure. This was obtained by turning the patient on the left side so that the free flowing mercury could carry the tube head from right to left.

pouch. On rare occasions if the gastric resection is performed so that the proximal loop is anastomosed to the greater curvature and the distal loop anastomosed to the lesser curvature at a higher point ambulation may result in the mercury bearing tube head passing into the proximal loop and being trapped there. In such cases the tube should be withdrawn and the patient turned on the right side in a semi sitting position. This will invariably result in the tube passing along the lesser curvature into the distal jejunal loop.

Fastening the tube to the side of the face can result in the small bowel plying itself upon the available tube so tightly that pressure necrosis with perforation of the bowel is produced. Instances of this have been reported. Letting the tube lie free results in a physiologic type of intubation since peristaltic activity often carries it downward in a physiologic fashion. In such cases tight fixation of the bowel upon the tube is prevented.



FIG 321 The balloon of the intestinal tube is dropped into the upper jejunum beyond the duodenojejunal flexure. Notice the acuity of the angle of the second and third limbs of the duodenum as shown by the kinking in the long tube.



FIG 322 Radiograph taken with the Cantor tube in the stomach and the patient lying flat on his back. Notice the position of the tube head in the left paravertebral gutter which is the most dependent portion of the stomach in this position.

Preparation of the Child Patient and Passage of a Tube in Children

- 1 Give the child a half grain of luminal one hour prior to passing the tube
- 2 Apply 1 per cent Pontocaine in 1 per cent ephedrine to the side of the nose through which the tube is to pass
- 3 Use the same technic for inserting the tube and passing it into the stomach as described for adults
- 4 The last two steps in the adult method of turning the patient flat on his back in Fowler's position and then turning the patient on the left side may be omitted in infants and children. Instead permit the child to move about freely and encourage him to stand. The relative simplicity in the anatomy of the stomach in the infant and child is such that intubation usually is accomplished quite easily.

Removal of the Long Intestinal Decompression Tube

- 1 Inject adults with $\frac{1}{8}$ grain of morphine sulfate and $\frac{1}{400}$ grain of atropine sulfate a half hour prior to removal of the tube
- 2 Remove the tube gently and firmly. Do not hurry. If there is any resistance to the removal of the tube wait until the spasm is released and then pull firmly.
- 3 Any tube whose head has passed into the colon should be removed from above. The only exceptions to this rule are those cases in which the tube head protrudes from the anus or is readily palpable in the rectum. In such cases for esthetic reasons it is best to pull the tube out from below.
- 4 No given period of time should be allowed to remove the tube. Most of the tubes can be removed in 5 to 10 minutes and some in less

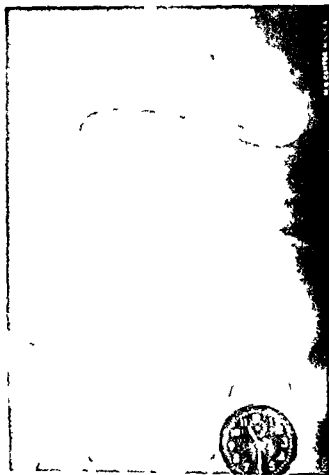


FIG. 319 With wing, the passage of the tube head through the pylorus and sitting the patient up result in the downward passage of the tube into the second limb of the duodenum. Note the direction of the second limb in the duodenum.

most direct downhill course from the stomach. In intubating a stomach of this type ambulation alone is sufficient to pass the tube rapidly through the pylorus without the necessity of any of the maneuvers described. An atonic stomach would require the described maneuvers if the pylorus were fixed at a high point. However, since the intubator usually does not know the type of stomach being intubated, it is a good idea to maneuver all patients as described. Then if a check film shows an unsuccessful intubation, one might suspect that a

J type of stomach was not present and ambulation could then be used. If the stomach being intubated is a postgastrectomy stomach ambulation is sufficient to pass the tube head down the distal loop because the outlet of the stomach is at the inferior portion of the vertically lying gastric



FIG. 320 The tube head is now at the duodenojejunal flexure. This was obtained by turning the patient on the left side so that the free flowing mercury could carry the tube head from right to left.

pouch. On rare occasions if the gastric resection is performed so that the proximal loop is anastomosed to the greater curvature and the distal loop anastomosed to the lesser curvature at a higher point, ambulation may result in the mercury-bearing tube head passing into the proximal loop and being trapped there. In such case the tube should be withdrawn and the patient turned on the right side in a semi-sitting position. This will invariably result in the tube passing along the lesser curvature into the distal jejunal loop.

Taping the tube to the side of the face can result in the small bowel plying itself upon the available tube so tightly that pressure necrosis with perforation of the bowel is produced. Instances of this have been reported. If the tube lies free results in a physiologic type of intubation since peristaltic activity often carries it downward in a physiologic fashion. In such cases the plication of the bowel upon the tube is prevented.



FIG 321 The balloon of the intestinal tube is dropped into the upper jejunum beyond the duodenojejunal flexure. Notice the acuity of the angle of the second and third limbs of the duodenum as shown by the kinking in the long tube.

Preparation of the Child Patient and Passage of a Tube in Children

- 1 Give the child a half gram of luminal one hour prior to passing the tube
- 2 Apply 1 per cent Pontocaine in 1 per cent ephedrine to the side of the nose through which the tube is to pass
- 3 Use the same technique for inserting the tube and passing it into the stomach as described for adults
- 4 The last two steps in the adult method, i.e. turning the patient flat on his back in Fowler's position and then turning the patient on the left side, may be omitted in infants and children. Instead permit the child to move about freely and encourage him to stand. The relative simplicity in the anatomy of the stomach in the infant and child is such that intubation usually is accomplished quite easily.



FIG 322 Radiograph taken with the Cantor tube in the stomach and the patient lying flat on his back. Notice the position of the tube head in the left paravertebral gutter which is the most dependent portion of the stomach in this position.

Removal of the Long Intestinal Decompression Tube

- 1 Inject adults with $\frac{1}{2}$ grain of morphine sulfate and $\frac{1}{400}$ grain of atropine sulfate a half hour prior to removal of the tube
- 2 Remove the tube gently and firmly. Do not hurry. If there is any resistance to the removal of the tube, wait until the spasm is released and then pull firmly.
- 3 Any tube whose head has passed into the colon should be removed from above. The only exceptions to this rule are those cases in which the tube head protrudes from the anus or is readily palpable in the rectum. In such cases, for esthetic reasons, it is best to pull the tube out from below.
- 4 No given period of time should be allowed to remove the tube. Most of the tubes can be removed in 5 to 10 minutes and some in less



FIG. 323 Notice the change in position of the tube head with the patient in the erect position. This is a radiograph of the same patient taken a few seconds after Figure 322.

than 2 minutes. However, any resistance in withdrawal should be interpreted as indicating either a retraining spasm or possibly a large knot in the tube. In such cases the use of relaxing drugs and a wait of one to two hours may be necessary. If despite all the methods the tube still cannot be removed from above, it may then be cut off at the nose and permitted to pass downward. In such cases it will always be excreted in 1 to 19 days *provided there is no obstruction distal to the end of the tube*. In any case in which obstruction distal to the tube is suspected the tube must not be permitted to pass downward for excretion. To do this is to invite complete obstruction by the coiling of the tube at the point of partial obstruction.

Care of the Tube after Removal

Remove the used balloon and discard it. Apply suction through the tube with soapy water or de-



FIG. 324 Notice the mercury bearing tube head trapped in the proximal loop because of incorrect positioning. In intubating any patient who has had a gastric resection it is desirable to know exactly where the proximal loop is in order to avoid this accident. Withdrawal and reinsertion of the tube and ambulation of the patient will result in successful intubation.

tergent until clear. Then suction through and thoroughly rinse with tap water, then rinse thoroughly with distilled water. Soak for 24 hours in zephiran aqueous 1:1000. Then rinse with tap water. Soak for 24 hours in rose water to remove all odors from the tube. Then suction with distilled water. Hang up to dry for 48 hours. Reservice with a new balloon. A new balloon must be used for each intubation. This method of preparation, although time-consuming, not only cleans and sterilizes the tube but also removes all odor. Iodo must not be used in the cleansing process. Small amounts of Iodo retained within the tube have been known to produce a fatal gastritis.

Do not permit organic solvents, such as alcohol, benzene or xylol, to come in contact with the tube, since the cement which holds the bag to the tube is soluble in organic solvents and the attachment of the bag to the tube will be weakened.



FIG. 325 A radiograph of a child's stomach. Notice the simple course which makes intubation relatively easy.

To attach a new balloon apply the special rubber cement an inch from the tip of the tube. Allow to stand for 10 to 15 minutes until thoroughly dry. Apply a second coat of cement to the tube and immediately slip the end of the tube into the balloon. A Vienna pattern nasal speculum or a small hemostat will dilate the end of the balloon and facilitate the introduction of the tube end into the balloon. Allow to dry for 12 hours after which time the tube is ready for use.

The above instructions apply to the use of the Cantor tube. The Harris tube and Koslow tube balloons are simply tied on with black silk.

SUCTION DEVICES AND THEIR USE IN BOWEL OBSTRUCTION

A long intestinal decompression tube far down the gastrointestinal tract will be of little or no value if the source of negative pressure applied to its other end is poor or not working at all. When the suctioning device is not functioning properly the apparatus is worse than useless because it can completely block the tube and prevent the in-

creased intraluminal pressure from forcing the intestinal contents into the tube.

There are four main types of negative pressure which are successfully employed in conjunction with the long intestinal decompression tube.

Siphonage Suction

The simplest form of siphonage drainage of the gastrointestinal tract consists of passing a long intestinal decompression tube as far as possible and then permitting the proximal end to drop over the side of the bed into a collecting bottle. When a 16 Fr. tube such as a simplified single lumen intestinal tube reaches the stomach there is an immediate outpouring of gastric contents. The force with which the gastric contents comes out of the tube is often so great that they literally spurt out because of the great increase of intra-gastric and intra-abdominal pressure found in any case of intestinal distention. The stomach empties rapidly merely because of the presence of the tube in that viscus and because of the elasticity of the gastric wall which resists distention. When the tube head leaves the stomach and passes down the gastrointestinal tract into the small bowel there

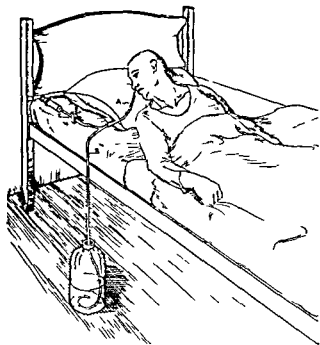


FIG. 326 Simplified siphonage unit for use in ambulatory patients.

is also a marked increase in intraluminal as well as intra abdominal pressure (acting upon the tube held in the small bowel) which forces the intestinal contents into the lumen of the tube and so out by siphonage. However, since the intestinal contents in any case of intestinal distention of moderate degree are a mixture of gas and liquid bubbles of gas enter the lumen of the tube with the liquid intestinal contents. When this occurs the siphonage is broken and does not become reestablished until the bubbles of gas are evacuated. To prevent this occurrence Leithrauser has devised a simplified priming device for siphonage in the treatment of intestinal distention.

One great advantage of this simple siphon method using a priming device is that it necessitates only a minimum of nursing care. There is no cumbersome equipment requiring constant attention and since Leithrauser is an ardent advocate of early ambulation this procedure can readily be adopted without disconnecting or removing any apparatus. The patient merely gets out of bed and walks carrying the tube with him. This simplified siphoning unit is depicted in Figure 326.

Continuous Suction by Water Displacement Method

This method of continuous suction drainage is one of the simplest and oldest methods still in use. It was used by Mats and Brissler early in the 20th century. Ward in 1925 again called attention to this method of suction drainage and modified the apparatus. The work and writing of Wangensteen and his co-workers provided the greatest impetus to the use of this method of obtaining negative pressure at the end of the intestinal tubes to suction out the gastro-intestinal tract. They modified the apparatus in an effort to so simplify it that it could be made available to any hospital and set up on very short notice. This apparatus is based upon the observation that if a bottle is filled full of water and connected with a second bottle by rubber tubing, which is airtight, the flow of water from the upper bottle to the lower one leaves a vacuum in the upper bottle which then acts as a negative pressure exerting a suctioning force which can be applied to the end of an intestinal tube. At the time this apparatus was introduced

the chief function was gastroduodenal suction to drain the upper gastro intestinal tract.

At present this simple method is also used as the suctioning force for the long intestinal decompression tube in the aspiration of the entire gastro intestinal tract. When this method is properly used and supervised by one who understands the mechanics involved excellent results are obtainable. Unfortunately many surgeons and nurses do not understand the mechanics of or the necessity for airtight equipment so that in many hospitals where results have been poor this method has fallen into disrepute. Our own experience with this method has been good. With proper supervision of the nursing staff and frequent checks on the equipment to be certain that it is properly hooked up and airtight as good results are possible with this equipment (and at a lower cost) as with any other available today. The apparatus consists of

- 1 Two large glass bottles of 4000-cc capacity
- 2 A two hole rubber stopper which fits one of the bottles tightly
- 3 A canvas bag to hold one of the bottles in an inverted position
- 4 A stand to hold the upper bottle 7 feet above the floor
- 5 About 16 feet of rubber tubing of $1\frac{1}{4}$ inch lumen
- 6 Two glass tubes 4 and 16 inches long respectively
- 7 Several screw clamps

To assemble the apparatus as simply as possible the canvas or wire sling is fitted over one of the bottles and this is filled with water. The glass tubes are so placed in the two holed rubber stopper that the longer glass tube extends almost to the bottom of the bottle. The shorter glass tube extends to a point just within the mouth of the bottle. The rubber stopper containing the two tubes is then fitted tightly into the mouth of the bottle to be inverted and hung upside down on the stand at a distance of 5 feet from the floor. The short glass tube is connected by a 6 foot piece of rubber tubing to a second bottle which rests on the floor and contains 200 to 300 cc. of water. The rubber tube must be below the water level in

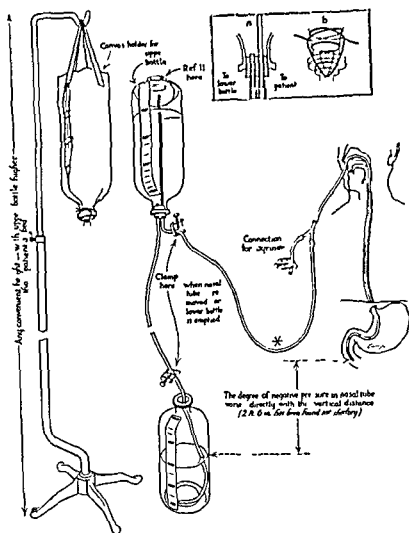


FIG. 327 Wangenstein type of negative pressure depending upon water displacement

the bottle on the floor. This leaves a vacuum in the upper bottle thus creating a negative suction pressure which is transmitted to the end of the intestinal tube. This is the basic mechanism utilized to produce a continuous negative suction pressure for decompression of the gastro intestinal tract. As time went on modifications to this simple setup were introduced by Wangenstein and other workers. One of the best of these modifications was the placing of a graduated collecting bottle between the intestinal decompression tube and the long glass tube which transmits the negative pressure. It is thus very easy to measure the amount of gas and material withdrawn while at the same time inserting a protecting mechanism in the event that the upper bottle is accidentally connected di-

rectly to the intestinal tube by its short glass rod just within the mouth of the water-containing upper bottle. Were this to happen water would run into the intestinal tube and into the stomach or bowel of the patient. In addition the stopgap bottle prevents the intestinal mucosa from being exposed directly to too great a suction from the upper bottle. This consideration is only theoretical since Paine demonstrated that a suction pressure of 1000 cc of water had no injurious effect upon the mucosa in contact with the holes in the intestinal tube.

The apparatus of Fritz is one of the newest commercially available systems utilizing this water displacement principle to create a vacuum. This is an automatic drainage and aspirating apparatus

capable of continuous suction. The safety feature is the elimination of all valves and cocks which might interfere with the proper operation of the equipment. There is a specifically designed rotary valve mounted between the bottles which automatically control the flow of water suction and pressure and which is closed except when the bottles are in a vertical position. An automatic spring lock engages and holds the movable parts of the machine in proper position. The bottle connection cannot be reversed which eliminates any

possibility of injecting air instead of withdrawing it. Once this apparatus is properly assembled and connected to the decompression tube the condition of the patient determines the time which should elapse between the turning of the bottles. If the machine stops functioning, the tube should be withdrawn a few inches then pressed again with rotation and the patient given some water to drink. When this is done the machine will usually begin to function again. Never use a clamp on the suction tube from the machine when the apparatus is in use. Air must escape freely from the end of the shaft opposite the suction end of the shaft.

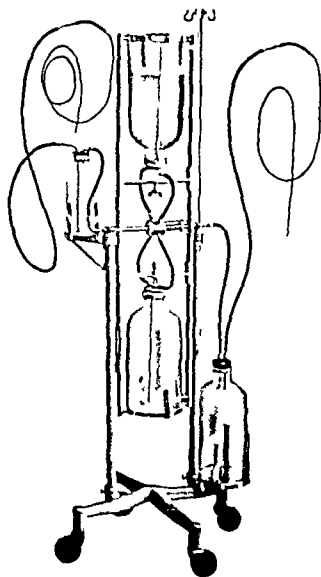


Fig. 128 Fritz type of suction unit. This is an efficient unit depending upon the water displacement method to produce a negative suction pressure.

Continuous Suction by Effect of Heat upon Air

The Gomeo thermotic drainage pump affords a mild precision-controlled intermittent suction which is applied to the end of the long intestinal tube. This apparatus is based upon the utilization of the expansion and contraction of air when subjected to variations in applied electric heat. The gas thermometer, where the expanding or contracting gas moves the indicator is the most familiar application of this principle. For each degree of temperature change above or below zero degrees centigrade there is a volume change of $\frac{1}{273}$. If this principle is used in a heating chamber of suitable size with the necessary heating unit, timing apparatus and directional valves a flow of air in one direction may be obtained. As a portion of the heated air is driven off to the atmosphere and the remaining air allowed to cool a relative vacuum exists with the result that air is drawn into the chamber through the suction inlet. The negative pressure is developed in the cylinder shows a gradual rise from zero degrees centigrade until it reaches its peak in 12 seconds whereupon it drops rather suddenly. When this drop occurs the thermostat again permits heating of the cylinder and at the end of five seconds the curve begins to rise again. Using a suction bottle as an intermediary the curve of negative pressure tends to flatten out. Because sound waves are very disturbing to the seriously ill patient thermotic pumps were studied from the point of view of acoustic measurements of the thermotic pump.

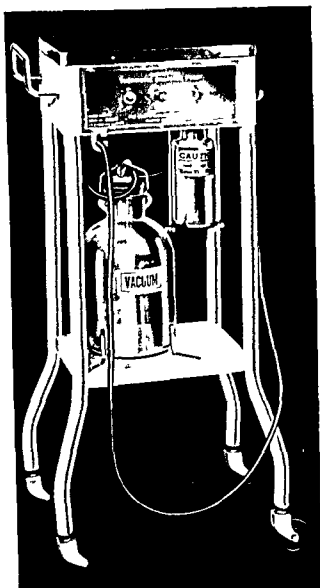


FIG 329 The Gomco Thermotic pump. This utilizes the contraction and expansion of air when subjected to variations in electrical heat.

in action demonstrated that the noise level of this device was extremely low. It was found by actual measurement that the ticking of an ordinary spring driven alarm clock was more audible than the noise produced by the pump. The degree of suction obtained ranges from 90 mm. of mercury negative pressure to a maximum of 120 mm. and is precision controlled. The pump may be operated without interruption for any length of time. There is no diminution of its drainage effectiveness with continued usage. Since the pump has

no motor or parts requiring attention a minimum of nursing care is required. A small pilot light is utilized to indicate when the pump is operating. It goes on and off intermittently. A one gallon suction bottle and a safety overflow bottle are arranged on the stand. We have found this equipment to be extremely effective.

Motor Pumps and the Stedman Pump

Motor driven pumps are frequently used to create a negative suction pressure to be applied to the end of a long intestinal decompression tube. By using a controlled low suction pressure and inserting a stopper bottle between the pump and the patient good results are obtainable. The only objection to motor driven pumps is that they are often noisy and cumbersome and in addition are often incapable of continuous operation without excessive wear.

The Stedman pump was developed to obviate the objections to the use of a motor pump as a source of negative pressure. This pump is activated by electromagnetic coils on the same principle as an electric meter. Since it has only three moving parts this diminutive suction pump is noiseless and vibrationless and is capable of running day and night. This equipment is small and readily portable. It can be plugged into any alternating outlet with the same rating as the pump. In using this apparatus it is essential that a collecting bottle be interposed between the patient and the pump. Beyond a weekly drop of oil on its bearings if it is being operated continuously this pump requires no attention.

RADIOLOGIC INTESTINAL INTUBATION

Mellins and Cantor reported a simple method for rapid intubation with the mercury tipped long intestinal decompression tube. The method is based upon the effect of the gastric peristaltic waves upon the mercury within the balloon of the intestinal tube. By noting the direction of flow of the mercury contained within the tube head it is possible to so position the patient fluoroscopically that the tube head will pass through the pylorus into the first limb of the duodenum in less than 15 minutes. From this point with further positioning the tube head may be rapidly passed down



FIG. 330 Notice the effect of peristalsis upon the balloon of the Cantor tube within the stomach. The tube has just proceeded to the pylorus and is halfway through. The demonstration of waves in the mercury in the tube head can be utilized by the radiologist to successfully pass tubes.

the second limb of the duodenum. The technique is as follows:

- 1 Pass the long intestinal decompression tube into the stomach.
- 2 Suction out the stomach and then send the patient to X-ray.
- 3 Place the patient under the fluoroscope and give him a drink of ice water. This results in fluoroscopically visible peristaltic waves in the mercury in the same direction as the peristaltic waves induced in the stomach. Intubation then simply involves positioning the patient fluoroscopically so that the mercury-tipped tube will always be directed in

the same direction as the waves. With this method intubation will take place within 15 to 30 minutes in well over 90 per cent of patients.

There are two underlying principles utilized in this radiologic method. First, the mercury in the balloon acts in the same manner as a barium bolus so that the fluoroscopist can determine the force and frequency of peristalsis and the direction of the pyloroduodenal canal. Second, the patient can be fluoroscopically positioned to place the mercury balloon just above the pylorus with the ice-cold water then given as a peristaltic stimulant.

SURGICAL INTUBATION

In those cases in which the orthodox methods of intubation are unsuccessful, Rodney Smith has proposed intestinal suction gastrostomy as a method of intubation. This is considered to be particularly indicated for those cases in which intubation by the usual methods is unsuccessful and where the condition of the patient is such that intestinal decompression is essential. His method is as follows:

- 1 A Ryle stomach tube is passed and the stomach is emptied and kept empty by suction throughout the operation.
- 2 A small right upper paramedian incision is made under local anesthesia which exposes the pylorus and pyloric antrum. A stab wound is made to the left upper rectum and a Miller Abbott tube with the balloon collapsed is threaded through it and drawn out through the main incision. The pyloric antrum is then drawn into the wound and a stab incision is made 1 inch from the pylorus.
- 3 Through this opening in the stomach a rigid rubber tube large enough to transmit the Miller Abbott tube is passed and pushed through the pylorus into the first portion of the duodenum.
- 4 The Miller Abbott tube is lubricated and a sufficient length is passed through the rigid rubber tube to carry the tip around to the duodenojejunal flexure. The rigid rubber tube, now on heating the Miller Abbott tube is withdrawn a sufficient distance to leave its end just inside the

stomach where it is fixed with a single catgut suture

6. Four fifths of this rigid rubber tube are now tunneled as in the performance of a Witzel gastrostomy
7. The stomach is then returned to the abdomen and the slack of the Miller Abbott tube inserted through the stab incision is withdrawn. Finally the remaining fifth of the rigid tube of rubber is brought out and anchored to the skin by a single suture. The main incision is now closed.
8. The balloon of the Miller Abbott tube is inflated and continuous suction applied to this tube. The Ryle tube is removed.

Devine proposed surgical intubation for these patients. The Devine method calls for the insertion of a Miller Abbott tube directly into the jejunum through a jejunostomy in which the tube is brought out through a small stab wound. Inflation of the balloon would then permit the tube to pass down the gastro intestinal tract while decompressing it.

Nolan and Finley proposed the simple method of threading the long intestinal decompression tube tipped with a mercury bearing balloon (Cantor) rapidly down the gastro intestinal tract at the time of surgery. This method was ideally suited to those cases in which the tube head was found to have passed the ligament of Treitz and less well suited to those cases in which the tube head was still in the stomach. Nolan and Finley however reported successful intubation even under these circumstances. We have found intubation difficult when the tube was found to be in the stomach or duodenum at the time of surgery. If the tube is found to have passed into the upper jejunum or beyond the ligament of Treitz surgery need not be delayed. When the abdomen is opened in such cases it is simple to thread the long intestinal tube down to the cecum rapidly while applying continuous suction to its proximal end. In a matter of 10 or 15 minutes the entire gastro intestinal tract proximal to the point of obstruction can be readily decompressed. To accomplish this it is sufficient merely to elevate the loop of bowel just proximal to the tube head. By so doing the highly labile heavy mercury tube head rapidly runs downhill into the distended distal bowel. Continu-

ous suction applied to the end of the tube assures rapid emptying of each loop of bowel so treated.

USE OF INTESTINAL DECOMPRESSION SOUND

In those cases in which immediate surgery is indicated and the tube head is still found to be in the stomach needless delay in surgical intervention may be avoided by the use of the intestinal decompression sound. This is especially useful in those cases of suspected or actual strangulation obstruction where sufficient time to assure successful intubation is not available and in which the distention is marked because of delay in diagnosis or in seeking treatment.

The use of the decompression sound to empty the bowel at the time of surgery is not new. Nicholas Senn in his treatise on intestinal surgery in 1889 advocated the correction of intestinal distention at the time of operation for bowel obstruction. To accomplish this he proposed that the bowel be opened and aspirated. In order to empty loops of bowel kinked by the weight of the intestinal contents he manually straightened them to encourage liquid material to flow toward the point of aspiration. In addition after the obstructive lesion had been corrected he advised the use of pressure dressings and saline cathartics to prevent recurrence of intestinal distention and stasis.

Monks as early as 1903 was an ardent proponent of this method of decompression. The method used by Monks consisted of threading the bowel onto a glass tube which permitted its emptying. Albert Ochsner used the method extensively in 1912. H. A. Gamble after observing Ochsner used the technique and wrote glowing accounts of its effectiveness. Holden in 1926 began to write voluminously and enthusiastically about the use of a trochar as a means of intestinal evacuation at the time of surgery in intestinal obstruction. Sweek and Patterson were impressed by the work of Holden and used this method with considerable success. For their cases they used an enterostomy tube threaded through the bowel.

However considerable criticism of this method of surgical decompression arose in the pre antibiotic era. From their observations Ochsner and Storck concluded that stripping should seldom be

employed because of its resultant lowering of blood pressure and inhibition of intestinal tonus due to trauma. They reported a mortality rate of 68.7 per cent for this method of decompression. Loven and Morton have both pointed out that shock could be produced by the stripping of the distended obstructed bowel during its process of plication onto the decompression sound. Wengenstein opposed this method, believing that enterotomy would accomplish essentially what this procedure professed to do and at a lesser risk. As a result of the wave of criticism the method fell into disrepute so that by 1930 it was rarely if ever used.

In recent years, with the widespread use of antibiotics, surgical decompression of the gastrointestinal tract has again come into being. The poor result obtained in the management of intestinal obstruction by delayed surgical intervention and the dependence upon the long intestinal decompression tube to deflate the bowel were also factors in this rebirth. We believe the method of decompression by sound at the time of surgery is useful in that small group of cases in which intestinal intubation is an successful and for which immediate surgery is indicated. In particular this method circumvents the need for enterostomy, whose field of usefulness in this era is extremely limited.

There are many different types of decompression sound proposed to accomplish intestinal deflation at the time of operation. Wengenstein recently advocated the use of a trochar of sufficient size to accommodate a 22 Fr. rectal tube. Williams and Williams were greatly impressed by the ease with which intestinal distended loops could be

emptied by the use of a Poole suction tip. Williams and Williams report that 6 feet of small bowel can thus be emptied and threaded on to the suction tip. They found a rectal tube unsatisfactory when used in the same way. Lowdon introduced a method of aspiration by the use of an aspirating needle with a diameter of 0.7 to 0.9 mm. Lowdon suggested that it is often advisable to a pirate at more than one point using a fresh needle for each puncture. He points out, however, that with his method only the gaseous content of the bowel can be removed since fluid blocks the needle so rapidly that no useful reduction of distention could be obtained.

Cantor introduced a simplified intestinal decompression sound by means of which the entire gastrointestinal tract may be decompressed if desired. For practical purposes the small bowel alone requires the use of this sound. The sound introduced by Cantor consists of three 12 inch sections of tube with a 20 Fr. lumen. The sections can be screwed into one another so as to make a 3 foot long sound if necessary. Practically more than 2 feet of sound are rarely if ever needed. The end of the sound which is to be used as a suction tip is rounded and presents a lumen. Just beyond the end of the tube three elliptically shaped holes are arranged spirally around the tube. These permit rapid aspiration with a minimum of plugging by intestinal particulate matter. One inch below the end of each section of sound an elliptic hole is found. The function of this hole is to permit an intermittent break in the suction applied to the bowel when the sound is inserted. When suction is to be applied to the bowel the index

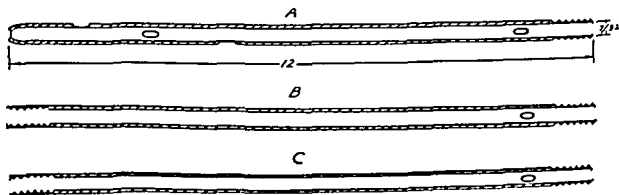


FIG. 331. Schematic drawing of Cantor's intestinal decompression sound.

finger of the hand holding the sound covers this hole. By so doing, the negative pressure will press down the sound and readily aspirate the intestinal contents into the end hole and the spirally arranged elliptic hole. When the loop of bowel is being threaded onto the sound and suction is not desired for the moment, simply raising the finger covering the make or break hole results in a loss of suction. One such make or break hole is found at the end of each section of the decompression sound to permit the use of each section as an independent unit.

The technique of using the intestinal decompression sound is simple. When the abdomen is opened the first loop of distended bowel is picked up. It is then well packed off by moist warm lap towels. A purse string of 000 chromic catgut is placed on its antimesenteric edge. A small opening is then made within this purse string loop and the decompression sound, *I* with continuous suction applied to its end at point *1* is rapidly inserted through the opening in the bowel. The purse string is then snugged. This prevents any leak of intestinal contents. The sound is then gently passed proximally along the loop of bowel emptying it. When this loop has been emptied it is gently threaded onto the sound and continuous suction is applied to the next proximal segment of bowel. When sound *I* has been completely threaded with collapsed bowel section *P* is screwed onto section *A* at point *1*. While this is being done suction is discontinued by merely elevating the finger from the make or break hole at the end of each section of sound. After joining sounds *A* and *B* together the hole which was used as a break in suction in section *A* is pushed into the lumen of the bowel where it acts as an aspirating hole. Similarly the index finger is then placed upon the hole at the distal end of the sound *B* just below point *2* when suction is desired and elevated when suction is no longer required.

After the proximally distended bowel has been decompressed the sound is slowly removed until only 1 inch beyond its tip remains within the lumen of the bowel. The direction of the sound is then reversed and the bowel distal to the loop selected for decompression is aspirated of its contents until the point of obstruction is reached. At this point

the intestinal decompression sound has served its purpose. It is now removed and the purse string tied. A second purse string of 000 silk is then placed about the first. The collapsed bowel may then be readily packed away from the site of obstruction which can be effectively dealt with.

By using this intestinal decompression sound the entire small bowel may be rapidly and effectively decompressed using only one small enterotomy opening which is closed securely by purse string suture. With average care to prevent soiling the danger of contamination of the peritoneal cavity is negligible. A liberal use of antibiotics postoperatively renders the procedure safe as well as effective.

Wound infection may occasionally occur if a break in technique has occurred. A change of gloves and instruments before closing the abdomen and adequate draping of the wound with proper packing of the opened loop of bowel will obviate this danger.

A note of caution is in order in the use of this instrument. Since the obstructed bowel is generally edematous and often friable the bowel should be gently threaded along the sound *after* the distended bowel has first been collapsed by suction. In no case should the sound be pushed forcibly along the gastro intestinal tract. To do so is to invite perforation of the thinned-out edematous bowel wall. In addition the proximal bowel must always be threaded onto the sound under direct vision to avoid pushing the sound too strongly against the bowel wall.

In summary the use of the intestinal decompression sound may be said to accomplish the following objectives:

- 1 It prevents the development of the so-called water hose kinks when the distended intestine is placed back into the peritoneal cavity.
- 2 It makes it possible to operate even in the presence of widespread intestinal distention.
- 3 The toxic contents of the entire small bowel may be removed at the time of surgery.
- 4 The amount of fluid lost to the body within the lumen of the small bowel can be measured as it is being removed and replaced in the immediate postoperative period.

- 5 The point of obstruction can easily be found when the bowel is collapsed
- 6 Closure of the abdomen is simplified and hence the danger of wound disruption is decreased
- 7 Better anesthesia results from intestinal decompression because diaphragmatic excursion is much freer

As a result of all these factors postoperative complications are actually diminished and surgery is made safer and easier

LENGTH OF INTESTINAL DECOMPRESSION TUBE REQUIRED FOR INTUBATION

There is considerable variation of opinion as to the amount of intestinal decompression tube that should be placed in cases of intestinal distention. It has long been known that the small bowel pleats itself along the length of the tube much like an accordion so that at times with the 6 foot mark at the nose the tube head has been observed to emerge through the anus. Because of this one surgeon recommends the use of only 4 feet of tubing whereas others recommend 5 or 6 feet. Ideally the length of intestinal tube should be that amount which causes the least interference with the normal physiology of the small bowel whose functions are motility, secretion and absorption.

Cantor studied the effect upon the small bowel of variations in the length of the intestinal decompression tube. This study was conducted along clinical as well as laboratory lines using tubes 4



FIG. 332 Note the effect of placing 22 feet of bowel on a 4 foot long tube. Note the tight plication. There was little or no peristaltic activity in the bowel so plicated.



FIG. 333 Note the effect of placing 22 feet of bowel on a 4 foot long tube. Note the tight plication. There was little or no peristaltic activity in the bowel so plicated.



FIG. 334 At operation note the very loose plication of the bowel upon a 10 foot long tube. Approximately 22 feet of small bowel are plicated up in this 10 foot tube. Peristalsis in the plicated loops is good.

6 and 10 feet long. The objectives of the study were to note the position of the tube head 48 hours after successful intubation as shown radiologically with each of the tube lengths used. Then at the time of surgery the degree of plication of the bowel upon each of these tubes was noted as were the presence and degree of peristaltic activity. The laboratory portion of the study consisted of threading the same tubes (4, 6 and 10 feet long) along the gastrointestinal tract of fresh cadavers. In this portion of the study only the degree of plication of the small bowel and the length of tube required to reach the ileocecal valve were noted.

The results of this study were

1. X-ray study of the abdomen showed all three tubes to be in the same area of the ileum after 48 hours. This would indicate that under the same conditions a 4, 6 and 10 foot tube would come to lie near the ileocecal valve at the end of 48 hours.
2. A comparison of 4, 6 and 10 foot tubes used for decompression purposes demonstrates that at surgery the 4 foot tube causes the tightest bowel plication, the greatest impairment of peristaltic activity and the greatest decrease in absorptive power. The tighter the plication of the bowel upon the tube, the greater the chances of intussusception. A 6-foot long tube appears to be far better than a 4 foot tube because the plication is much looser and peristaltic activity is less impaired. The 10 foot long tube produces the least interference with the normal physiology of the bowel. Peristaltic activity is unimpaired and the maximum area of mucosa is available for absorption and secretion because the plication of the bowel on the tube is minimal. Intussusception is least likely to occur with this length of tube.

For all practical purposes there is little point in passing more than 6 feet of tube down the gastrointestinal tract if the bowel will not accommodate itself to this length of tube of its own accord. To pass more tubing without permitting peristalsis to carry it down results in looping of the tube within the bowel. This may cause knot formation in the tube. Emphasis is placed upon the point that the

intestinal tube must not be fastened to the face of the patient. By leaving the tube free the swallowing acts of the patient, ambulation and the return of peristaltic activity usually result in the tube being slowly pulled down the gastrointestinal tract without being pushed by the surgeon. Such conditions are ideal because the bowel will not pull down more tubing than it can accommodate. A dependence upon the normal physiologic mechanism by which foreign bodies pass along the gastrointestinal tract produces the least interference with this mechanism and results in looseness of plication of the bowel upon the tubes. When the tube is fastened to the face, the pull of peristaltic activity upon the tube head meeting resistance from the fastened tube results in tight plication. The degree of this plication depends upon how short a section of tubing is present within the bowel. The shorter the length of tube, the tighter is the plication. Ulceration due to pressure necrosis



FIG. 335 Note the effect of forcibly passing too much tubing down the gastrointestinal tract. Note the coiling of the tube far down the bowel. This predisposes to knot formation.

may occur in the mucosa of the bowel so tightly plicated followed by perforation with resultant peritonitis

INDICATIONS FOR USE OF INTESTINAL DECOMPRESSION TUBE

Indications for the use of intestinal decompression tube may be divided into two large groups. In the first are disorders of the small bowel and in the second disorders of the colon.

Small Bowel Disorders

Small bowel disorders requiring intestinal intubation may be divided into four main groups:

- 1 All cases of ileus due to atony of the bowel where the intestinal distention is not in any way the result of a mechanical obstruction
- 2 All cases of intestinal distention as a result of inflammatory intra abdominal processes
- 3 All cases of ileus due to mechanical lesions regardless of cause
- 4 Patients subjected to elective surgery who would usually require enterostomy or colostomy prior to surgery

Atonic Ileus Group All those individuals in whom the intestinal distention is not the result of any mechanical obstructive process may be placed in this group. In the early 1900's most of these patients were treated by enterostomy of one type or another. Loney in writing on this subject noted that in many cases subjected to enterostomy there was little escape of gas and that vomiting continued and the distention remained. Consequently he suggested jejunostomy as a preferable method. With the introduction of the long intestinal decompression tube this method fell into disrepute.

Those cases of postoperative distention in which the distention is not due to surgical trauma but rather to either local or diffuse peritonitis require the use of the long intestinal decompression tube. In these cases the bowel is filled with fluid containing particulate matter as well as gas. Because of this the use of a long decompression tube is essential if satisfactory results are to be obtained.

Retroperitoneal lesions are a notorious cause of paralytic ileus. In many of these cases the intestinal distention is severe and must be adequately



FIG. 336 The effect of colonic distention upon the position of the tube in the gastric fundus. Note that the direction of the tube simulates that found in the post gastrectomy stomach.

treated if the harmful effects associated with distention are to be avoided. The treatment of the primary disease however must be carried on independently. All such cases of paralytic ileus may be adequately treated by intestinal intubation with the long tube. Among the lesions associated with retroperitoneal causes of paralytic ileus is aneurysm of the abdominal aorta with leakage of blood into the retroperitoneal tissues. A tremendous intestinal distention is often present. The use of the long intestinal tube in such cases is only palliative since the primary lesion may not lend itself to surgical correction. In recent years the use of aortic grafts may salvage some of these hitherto hopeless cases. A completely decompressed bowel is a necessity if such definitive treatment is to be tried.

Diseases of the kidney, ureter or bladder are not infrequently associated with paralytic ileus. On occasion following cystoscopy for diagnosis a severe type of intestinal distention may super-

vene. Intestinal decompression by means of the long intestinal decompression tube adequately controls the intestinal distention in such cases. Retroperitoneal tumors not infrequently cause the development of a paralytic ileus preoperatively and particularly postoperatively. In this type of case the use of the long tube preoperatively serves a dual function: it not only decompresses the bowel so that conditions at surgery are improved but it also permits a far better exposure of the operative field by virtue of the plication of the small bowel upon the intestinal decompression tube. Permitting the tube to remain in place following surgery prevents the postoperative distention so often associated with this sort of surgery.

The gastro-intestinal atony often present in the aged may cause a severe degree of intestinal distention as a result of infection or trauma. Since the gastro-intestinal musculature is atonic the passage of a long intestinal decompression tube requires considerable patience and know-how on the part of the intubator. Because peristaltic activity is impaired one must depend upon the technique of intubation itself and the selection of the proper tube is very important. Since the Miller Abbott and Johnston tubes depend on the action of peristalsis upon an inflated balloon for their downward passage it would seem that they are not too well adapted for use in cases of this type. The simplified long intestinal tube (Cantor) is especially well suited for use in these cases because ambulation of elderly patients is desirable. The Cantor tube depending solely upon a free flow of mercury in a loose sac at the end of the tube changes position as a result of movement of this tube head.

Inflammatory Distention. Many patients requiring intestinal intubation fall into this group. Although the results obtained by the use of the decompression tube alone are usually highly gratifying, there are some patients in this group who require surgical intervention. The use of antibiotics in addition to the long intestinal decompression tube plus a working knowledge of electrolyte and water balance have produced a remarkable reduction in the mortality rate for this sort of case.

Included in this group is any patient in whom

the causative factor for the paralytic ileus is an inflammatory process within the abdominal cavity. This may be the result of a generalized peritonitis, local peritonitis or an abscess anywhere within the peritoneal cavity. Intestinal distention formerly a serious problem in the treatment of these patients is now eliminated by introducing a long intestinal decompression tube far down into the gastro-intestinal tract.

Localized peritonitis like that which occurs in pelvic peritonitis with tubo-ovarian abscess constitutes an individual problem in intubation. In these cases two types of ileus may be present—a reflex paralytic ileus as a result of infection in the peritoneal cavity or a mechanical obstruction caused by the adhesion of a loop of bowel to the inflammatory mass. The resultant edema in the bowel wall or kinking of a loop so adherent may cause mechanical obstruction. Although intestinal intubation in cases of this type can constitute the only method of treatment required, some patients may need surgical intervention to correct the mechanical obstruction. When the long tube has passed far down into the ileum and the bowel is decompressed there is an opportunity for the bowel wall edema to subside, releasing the mechanical obstruction. If the obstruction is due solely to functional causes, gas and liquid stool are usually passed per rectum by the fifth day. An effort should be made to permit the inflammatory process to subside with conservative measures. A waiting period of 10 days is not too long provided the temperature decreases, the white blood count returns to normal, intestinal decompression is maintained and the patient shows clinical improvement. During this time intravenous alimentation and a high caloric diet may be given by mouth. With the tube head well down the ileum, some utilization of the ingested food will occur.

Following hysterectomy intestinal distention may appear. In such cases two etiologic factors may be found:

- 1 A paralytic ileus due to localized infection in the cervical stump or vaginal vault or as a result of surgical trauma.
- 2 Mechanical obstruction due to adherence of a loop of bowel to the site of surgery.

In such cases intubation constitutes only one phase of the treatment. A careful search should be made to find the precipitating cause.

Before the intestinal decompression tube is removed from any patient in this group it should be established that the patient is not mechanically obstructed. This information may be obtained in several ways:

1. A small amount of dilute barium may be injected through the tube and its downward course checked radiologically. If the barium is shown to pass into the colon without producing puddling in the small bowel the continuity of the small bowel is established.
2. Passage of gas or stool by the patient after the tube has been clamped off is presumptive evidence of patency of the gastro intestinal tract.

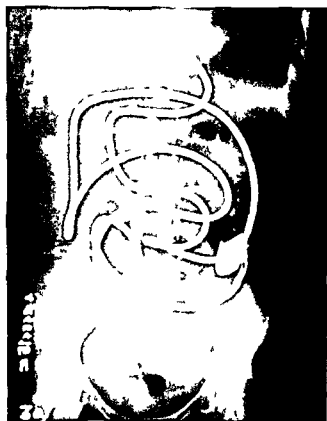


FIG. 337. Early post-operative intestinal obstruction as a result of a plastic exulcer. Note the small bowel distention. There is little interference as yet with peristaltic activity as long as the long tube readily passes down the gastro-intestinal tract.

Mechanical Obstructive Processes. This group is composed of those individuals in whom the intestinal distention is caused by a mechanical interference with normal peristalsis. This may be due to obstruction of the bowel from within its lumen from its wall from outside the lumen or as a result of interference with the circulation to the bowel. The lesions range from adhesive bands producing intestinal obstruction to mesenteric thrombosis.

We believe that the only function of the long intestinal decompression tube in such cases is that it allows the surgeon time to adequately prepare the patient for surgery or to better prepare the operative field. The only exceptions to this rule are those cases subjected to multiple operations with the formation of extensive adhesions. In these patients so many loops of bowel are adherent to each other and to the abdominal wall or other viscera that operative intervention with the possible exception of the Noble plication procedure is of little value. In these cases intestinal intubation may be utilized to carry the patient over the period of acute emergency to permit adequate pre-operative preparation for the formidable surgical procedure. Except for those occasions when the plication procedure is used successfully such patients are subject to repeated attacks of bowel obstruction by adhesions regardless of the surgery performed.

Intestinal intubation with the long tube often results in rapid and complete decompression in cases of small bowel obstruction caused by a post-operative adhesive band. Gas may be passed per rectum and the bowels may move. Many surgeons then discharge such patients as cured by intubation alone. Reports from many centers as well as our own experience reveal the startling fact that between 25 and 30 per cent of the patients return within one year with a second complete obstruction. In a review of small bowel obstruction at Grace Hospital over a 10-year period 58 patients were treated by intubation alone as the sole method of treatment without any study to determine the cause of the obstruction. Of this group 15 (26 per cent) returned to the hospital within one year and were again treated by intubation. There were even deaths. The 47 per cent mortality in this

small group is food for thought. From these experiences we believe that the long tube should never be removed until adequate radiologic studies have been carried out to determine the cause of the obstruction. We call attention to the fact that the use of the long tube merely relieves the intestinal distention which in turn releases the angulated or kinked bowel but does not interfere with the adhesive band causing the obstruction. If the band is allowed to remain it will be able to produce obstruction again when conditions are favorable. For this reason we believe that in any case in which a diagnosis of obstruction due to an adhesive band has been made radiologically, surgical correction is indicated even after decompression has been successfully obtained. In these cases the tube head will be often found to be just proximal to the point of obstruction. This simplifies the surgery.

In summary, it may be said that all cases of mechanical obstruction with the exception of cases presenting multiple and extensive adhesions from many previous operations, cases of inflammatory obstruction and cases of obstruction due to regional enteritis must be operated upon regardless of the etiologic factor involved. Intubation alone will not cure any of these cases. It may reestablish the continuity of the intestinal stream for the time being but this only lulls us into a false sense of security since a high percentage of patients so treated become obstructed at a later time. Should this occur in an area of the country where facilities are limited or should some other major pathologic process appear at the same time as the recurrence of the obstruction, the life of the patient may be needlessly lost.

Prior to Elective Surgery (Nonsurgical Enterostomy) Formerly preliminary enterostomy was required in cases where elective surgery was performed for known small bowel lesions. In any such case long intestinal tube intubation now obviates the necessity for enterostomy. The risk to the patient is thus reduced for two reasons. First the operative risk of enterostomy is removed and second plication of the small bowel upon the long tube simplifies the operative procedure by rendering the operative site more accessible.

Lesions of the Colon

The use of the long intestinal decompression tube for obstructive lesions of the colon appears to be controversial. Much of the confusion centering around the use of the long tube in the management of colonic lesions can be dispersed if we bear in mind the fact that the physiologic differences between these two portions of the gastrointestinal tract are such that a completely different surgical approach is required. Thus although the long tube is of value in obstructions of the right colon—especially in those cases in which the ileocecal valve is incompetent—the use of this tube in obstructions of the left colon is limited.

Right Colonic Lesions Obstructive lesions of the right colon particularly those of the cecum, the ileocecal valve and the right colon associated with an incompetent ileocecal valve lend themselves to decompression by the long intestinal decompression tube. In such cases the passage of the intestinal decompression tube down to the cecum



FIG. 338 Note the use of the long intestinal decompression tube in obstructions of the right colon associated with an incompetent ileocecal valve. Note the presence of the tube head in the ascending colon.

effectively decompresses the bowel proximal to the site of obstruction. In the presence of the commonly found high grade partial obstruction of the right colon peristaltic activity is vigorous. As a result the passage of the long tube is rapid. It is not unusual in such cases to find the tube head in the cecum or ascending colon. The liquid content of this portion of the gastro-intestinal tract readily permits decompression by a single lumen intestinal decompression tube of adequate caliber. With the proximal bowel decompressed in this manner primary resection and anastomosis can readily be accomplished after adequate preparation of the bowel with antibiotics.

The value of the long tube decreases as the obstruction moves away from the cecum. Therefore considerable judgement must be used in intubating obstruction of the hepatic flexure and transverse colon. In the presence of obstructive lesions in this portion of the right colon the competence of the ileocecal valve is of the greatest importance in



FIG. 340 The use of the long intestinal decompression tube to decompress the small bowel in obstructive lesions of the colon. Note how effectively the long tube decompresses the small bowel up to the ileocecal valve.



FIG. 339 Note the use of the long intestinal decompression tube in high grade partial obstructions of the right colon. The tube head has come to lie in the ascending colon.

deciding whether to use the long tube. If the ileocecal valve is incompetent long tube intubation is quite satisfactory because the ileum and in some cases the lower right colon may be decompressed. Right colonic decompression is possible because of the liquid content of this portion of the large bowel. Although the right colon can be satisfactorily decompressed by the use of the long tube in such cases it cannot be defunctionized. Despite this however a decompressed and clean right colon can be resected. In those cases in which the right colonic obstruction is associated with a competent ileocecal valve the long tube should never be used to decompress the right colon. Decompression of the small bowel is the best that one can hope for in such cases. If colonic decompression is required because of marked cecal distention some type of ceco-tomy is necessary. In such cases the long tube may be used in addition

to the cecostomy for the first few days until the cecostomy begins to function. By this time the small bowel will have been decompressed. The long tube should then be removed.

Obstructions of the right colon caused by cecal volvulus are closed loop obstructions. In this type of case the long tube should never be used except possibly as an adjunct to surgical correction.

Left Colonic Lesions The long intestinal decompression tube has a very limited use in obstructions of the left colon. Just as the left colon is physiologically different from the right colon and requires a different surgical attack, so also the problem of intestinal intubation differs in lesions of the left colon. Since the normal content of the left colon consists of solid fecal material it does not lend itself to decompression from above.

Although the long intestinal decompression tube will not decompress or defunctionize obstructive lesions of the left colon, it does have a place in

the management of such lesions. Patients are often admitted to the hospital with tremendously distended abdomens and no specific diagnosis. Such patients are usually in poor condition, dehydrated and anemic with poor plasma levels and electrolyte imbalance. In such cases the long tube may be used to advantage during the time the patient is being studied and prepared for surgery. In this fashion intestinal distention can be adequately controlled, giving the surgeon time to make a specific diagnosis and to prepare the patient for surgery. On a survey film of the abdomen these patients invariably present a marked large and small bowel distention suggesting the presence of an incompetent ileocecal valve. Once a diagnosis of carcinoma of the sigmoid with obstruction has been made a defunctionizing transverse colostomy is then indicated prior to resection. Under no circumstances should one attempt to use the long intestinal decompression tube as a defunctionizing instrument in such cases.



FIG 341 A lateral view of a patient with obstruction of the rectosigmoid. Note the tremendous intestinal distention typical of obstructions of the rectosigmoid.



FIG 342 The same patient as in Figure 341—obstruction of the rectosigmoid. This type of obstruction requires defunctionizing transverse colostomy.

The long tube can be profitably used in cases of diverticulitis of the left colon with obstruction in the presence of an incompetent ileocecal valve. In such cases keeping the small bowel decompressed allows sufficient time to treat the diverticulitis conservatively. This lesion tends to subside generally resulting in a reestablishment of the continuity of the gastrointestinal tract. However the presence of a competent ileocecal valve is a contra indication to the use of the long tube in this fashion. In such cases if the colon is completely obstructed a transverse defunctionizing colostomy is indicated.

Volvulus is a not uncommon obstruction of the left colon. In this case both the use of the long tube and defunctionizing colostomy are definitely contra indicated. Nothing less than surgical correction or the method of Braunford should be considered. The presence of a long tube in the ileum however will keep the small bowel decompressed following surgical intervention in such

cases. This is particularly of importance if resection of the colon is required.

Patients beyond the age of 60 who require resection of the left colon should be intubated prior to surgery. In this age group atony of the small bowel is common. When this occurs postoperatively it will be found that defunctionizing colostomy is of little or no value. These elderly debilitated patients whose condition is further deteriorated as a result of the operative procedure usually become difficult subjects to intubate. For this reason it is far better to pass the long tube several days pre-operatively and see that it is well down into the small bowel before the colon is resected.

Generally intubation in the presence of a mechanical obstruction of the left colon is relatively easy. This is true because most patients with mechanical obstructions of the left colon have active peristalsis so that the downward course of the long tube is apt to be rapid. Generally the tube head will be found in the terminal ileum within 24 hours. It is not unusual to find it at the ileocecal valve or in the colon at the end of 48 hours.

CONTRA INDICATIONS TO THE USE OF THE LONG INTESTINAL DECOMPRESSION TUBE

The use of the long intestinal decompression tube is contra indicated as a definitive treatment for small bowel volvulus intussusception internal herniation or any other type of mechanical obstruction—except as a preliminary to surgery. In the case of volvulus or intussusception too much time should not be taken to pass the tube. The well known tendency of such obstructions to strangulate must constantly be borne in mind.

Obstructions of the right colon at or near the hepatic flexure, associated with a competent ileocecal valve are definite contra indications to prolonged intestinal intubation. Volvulus of the cecum and right colon constitutes an absolute contra indication to intestinal intubation. Immediate surgical intervention is obligatory in this type of case.

All mechanical obstructions of the left colon are contra indications to the use of the long intestinal decompression tube as a colonic decompressing instrument or as a defunctionizing instrument. In



FIG. 343. Volvulus of the sigmoid colon. In this type of case immediate surgery is indicated. It is an error in technique to attempt to decompress the bowel by the long tube.

addition sigmoid volvulus is an absolute contraindication not only to the use of the long intestinal tube as a decompressing tool but also to defunctionizing colostomy as a defunctionizing form of treatment.

COMPLICATIONS DUE TO THE USE OF THE LONG INTESTINAL DECOMPRESSION TUBE

There are many complications to the use of the intestinal decompression tube. Almost every portion of the nasopharyngo-esophago-gastro-intestinal tract with which the long tube comes in contact has at one time or another been the source of some complication to intubation.

Nasal hemorrhage is not uncommon as a result of the passage of the long intestinal tube. Although this is more common with the metal tipped tubes it can occur with any type of long tube even the balloon tipped ones. The most common cause for this complication is the presence of some degree of nasal pathology. The intubation of infants is not infrequently associated with such bleeding.

A failure to keep the tube well lubricated and the mucosa of the nasopharynx moist may result in the development of otitis media due to irritation in the region of the ostia of the Eustachian tubes. To prevent this complication the patient should be instructed to sit up in bed with his head well forward. The ante-flexion of the neck helps to keep the tube away from the pharynx.

Trauma to the arytenoid cartilage at the time of insertion or as a result of prolonged use of the tube may lead to chondritis. Cases of ulceration of the larynx with the development of stenosis have been reported. Traumatic edema of the larynx produced by the use of the Miller Abbott tube has been reported by Kaufman, Serpico and Mersheimer. Morrison described a case of cricoid chondritis as a result of prolonged intubation. Wangensteen reported two cases in which the arytenoid cartilages were injured in the same fashion. Iglauer and Molt reported 10 cases of injury to the larynx due to the presence of the decompression tube. Eight of these cases developed laryngeal stenosis. Caruolo reported a case in which the patient lost his voice for two months after intubation. This was caused by the forcible passage of the Miller Abbott tube in a patient who

was coughing and gagging. Examination of the cords by an otolaryngologist revealed the vocal cords to be edematous without any other visible sign of trauma.

The presence of esophageal varices complicates the problem of intubation. Serious and profuse hemorrhage due to erosion of the long tube through such varices has been reported. Chaffee reported a case of this type in which the long tube had been present in the esophagus for seven days. The patient vomited considerable blood and died 36 hours later. At autopsy an erosion of an esophageal varix with ulceration and necrosis of the mucosa overlying at least two of the veins was found. Vinson reported benign stricture formation in the lower esophagus as a result of prolonged intubation. Superficial ulcerations which developed along the course of the long tube have been found at autopsy in the esophagus of patients subjected to prolonged intubation. The most common sites for such ulcers are at the points of anatomic narrowing.

One of the earliest unusual experiences with the long tube was reported by Pool. In this case a gastro-enterostomy had been previously performed. The long tube was found to have passed through the pylorus through the duodenum and proximal jejunal loop and then instead of passing down the distal jejunal loop it had passed back into the stomach through the gastro-enterostomy. From the stomach it again made a complete circle in the same fashion. Three such loops were made. As a result it was impossible to remove the tube without surgical intervention. A similar complication following intubation in a patient who had a gastro-enterostomy was reported by Cohen and Silverstein. A failure to recognize this sort of complication may result in mechanical damage to the intubated bowel if vigorous withdrawal is attempted.

The formation of knots in a long tube is usually the result either of initially passing too much tubing into the stomach or of passing it too rapidly. Occasionally the head of the long tube rapidly reaches the point of obstruction and becomes arrested at this point. The passage of more tubing into the bowel then results in a looping of the tube just proximal to the point of obstruction. This sets



FIG. 34a Notice the presence of a knot at the terminal end of the Miller Abbott tube. This completely stops the flow of intestinal contents through the tube.

the stage for a knot formation. When such knots occur in a double lumen tube, the tube must be removed because the suction holes are found distal to the point of knotting. In the use of a single lumen tube such as the Cantor tube, the development of such knots does not require the removal of the tube. Being a single lumen tube, additional holes may be cut in the tube and additional tubing permitted to pass downward into the bowel. Decompression can then be obtained. In the removal of such knotted tubes, the knotted portion of the tube must be drawn out through the mouth. It is not rare to find a mass of knots measuring 2 to 3 cm. in diameter so that the tube cannot be pulled back through the nose. Such knot formation has been described with all types of intestinal tubes. Caruolo reported the formation of a knot 6 cm. in diameter in a long tube. Many authors, among them Frenizer, McKittrick and Sarris, and Him-

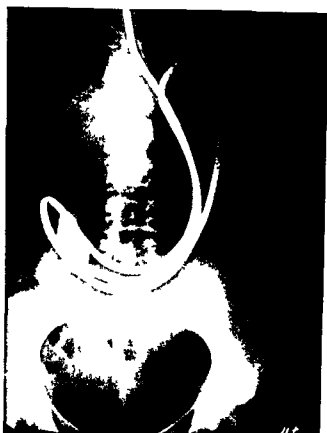


FIG. 34b An example of effect of passing too much tubing into the stomach initially. Notice the looping of the tube impeding its downward passage.

son have reported cases in which this accident occurred.

In a small percentage of cases in which the intestinal decompression tube is permitted to remain in the gastro intestinal tract for a period of time, it may be found that the balloon tipped tubes take up gas into the balloon. Because of this, it may be found that the balloon is more inflated than it should be or gas may be found in the balloon of a tube when none should be found. This accident can easily be discovered by radiologic study prior to the removal of the tube. It is quite characteristic for the mercury in the gas filled balloon to assume a circular form approximately the size and shape of a quarter. The presence of this complication may be serious. If a balloon containing gas is found to have progressed distal to the site of an anastomosis, attempts at withdrawal of the tube may cause a tear in the anastomosis with a resultant peritonitis. In a case of this type, the surgeon has two alternatives. He can cut off the tube



FIG 346 Note the accumulation of gas in the balloon of the intestinal decompression tube. Note also that the mercury is compressed in the shape of a silver coin.



FIG 347 The same patient as in Figure 346, note the injection of dilute barium puddled at the point of obstruction.

at the nose and permit it to be excreted per rectum or he can open the abdomen and compress the bowel containing the inflated balloon between his hands. The rupture of the balloon readily permits an easy removal from above. In no case should an enterotomy be performed to remove the inflated balloon. If the surgeon elects to permit the balloon to pass out per rectum, he must observe the patient carefully. It is essential to be certain that the inflated balloon tipped tube does not obstruct the bowel. It is often possible to cause deflation of the gas-filled balloon by the simple expedient of passing a second tube which decompresses the bowel near the distended balloon. As a result of the decrease in intestinal distention at the site of the inflated balloon, the gas pressures will be altered. Now the pressure of the gas within the balloon will be greater than that in the lumen of the bowel. The law of diffusion of gas is such that the gas would tend to leave the balloon and pass into the

lumen of the bowel from whence it would be removed. This is particularly apt to occur if the balloon is distended by carbon dioxide or hydrogen sulfide gas. These are rapidly diffusible through rubber membranes. If the distending gas is nitrogen, its speed of diffusion through rubber is very slow. Weeks may elapse before this gas completely diffuses from the balloon. For this reason too much time should not be permitted to elapse before deciding upon a course of action. This subject is completely reviewed in the chapter dealing with intestinal distention (Chapter 17).

An unusual complication in the use of the Miller Abbott tube was reported by Wilkie. In this case occlusion of the tube lumen was produced by a shrinking of the thread used to tie the balloon to the tube at its proximal end. As a result following the introduction of the tube into the small bowel, neither inflation nor deflation was possible. The balloon contained 10 cc of air. Attempts at removal of the tube failed. It was impossible to withdraw it beyond the duodenojejunal



FIG. 34a Notice the presence of a knot at the terminal end of the Miller Abbott tube. This completely stops the flow of intestinal contents through the tube.



FIG. 34b An example of effect of passing too much tubing into the stomach initially. Notice the looping of the tube impeding its downward passage.

the stage for a knot formation. When such knots occur in a double lumen tube the tube must be removed because the suction holes are found distal to the point of knotting. In the use of a single lumen tube such as the Cantor tube the development of such knots does not require the removal of the tube. Being a single lumen tube additional holes may be cut in the tube and additional tubing permitted to pass downward into the bowel. Decompression can then be obtained. In the removal of such knotted tubes the knotted portion of the tube must be drawn out through the mouth. It is not rare to find a mass of knots measuring 2 to 3 cm. in diameter so that the tube cannot be pulled back through the nose. Such knot formation has been described with all types of intestinal tubes. Carullo reported the formation of a knot 6 cm. in diameter in a long tube. Many authors among them Frenizer, McKittrick and Sarri, and Hin-

son have reported cases in which this accident occurred.

In a small percentage of cases in which the intestinal decompression tube is permitted to remain in the gastrointestinal tract for a period of time it may be found that the balloon tipped tubes take up gas into the balloon. Because of this it may be found that the balloon is more inflated than it should be or gas may be found in the balloon of a tube when none should be found. This accident can easily be discovered by radiologic study prior to the removal of the tube. It is quite characteristic for the mercury in the gas filled balloon to assume a circular form approximately the size and shape of a quarter. The presence of this complication may be serious. If a balloon containing gas is found to have progressed distal to the site of an anastomosis attempts at withdrawal of the tube may cause a tear in the anastomosis with a resultant peritonitis. In a case of this type the surgeon has two alternatives. He can cut off the tube



FIG. 346 Note the accumulation of gas in the balloon of the intestinal decompression tube. Note also that the mercury is compressed in the shape of a silver coin.



FIG. 347 The same patient as in Figure 346; note the injection of dilute barium puddled at the point of obstruction.

at the nose and permit it to be exerted per rectum or he can open the abdomen and compress the bowel containing the inflated balloon between his hands. The rupture of the balloon readily permits an easy removal from above. In no case should an enterotomy be performed to remove the inflated balloon. If the surgeon elects to permit the balloon to pass out per rectum, he must observe the patient carefully. It is essential to be certain that the inflated balloon-tipped tube does not obstruct the bowel. It is often possible to cause deflation of the gas-filled balloon by the simple expedient of passing a second tube which decompresses the bowel near the distended balloon. As a result of the decrease in intestinal distention at the site of the inflated balloon, the gas pressures will be altered. Now the pressure of the gas within the balloon will be greater than that in the lumen of the bowel. The law of diffusion of gas is such that the gas would tend to leave the balloon and pass into the

lumen of the bowel from whence it would be removed. This is particularly apt to occur if the balloon is distended by carbon dioxide or hydrogen sulfide gas. These are rapidly diffusible through rubber membranes. If the distending gas is nitrogen, its speed of diffusion through rubber is very slow. Weeks may elapse before this gas completely diffuses from the balloon. For this reason too much time should not be permitted to elapse before deciding upon a course of action. This subject is completely reviewed in the chapter dealing with intestinal distention (Chapter 17).

An unusual complication in the use of the Miller-Abbott tube was reported by Walkie. In this case occlusion of the tube lumen was produced by a shrinking of the thread used to tie the balloon to the tube at its proximal end. As a result following the introduction of the tube into the small bowel, neither inflation nor deflation was possible. The balloon contained 10 cc of air. Attempts at removal of the tube failed. It was impossible to withdraw it beyond the duodenojejunal

flexure where it had become arrested. The tube was cut off at the nose and permitted to be excreted per rectum. This took three days. Examination of the tube after its removal disclosed the cause of its arrest to be the presence of excessive balloon proximal to the tie. This permitted the formation of a projection around the proximal tie of sufficient size to be gripped by the bowel in spasm. Schlucke, Bergen and Dixon report collapse and general peritonitis following partial removal of the Miller Abbott tube when the balloon could not be deflated. Wangensteen reported a case in which the balloon of the Miller Abbott tube had passed halfway through the ileocecal valve into the cecum with the other half remaining in the ileum. The tube could not be withdrawn nor could the portion of the the balloon beyond the ileocecal valve be deflated. Finally the balloon ruptured spontaneously permitting its withdrawal. Harris and Gordon speak of their inability to remove a Miller-Abbott tube containing mercury after it had passed through the ileocecal valve. Persistent attempts to pull the bag through the ileocecal valve resulted in a tear of the bowel with the formation of an abscess. Kaplan and Michel report a case in which 70 cc. of air were found in the balloon of an intestinal tube which had passed into the terminal ileum.

Tolley reported that the pulling of the bowel upon a long tube may cause ulceration of the mucosa and perforation. This is apt to occur if the balloon is left inflated and the tube is fastened to the nose. Under these conditions the bowel becomes very tightly pleated on the tube and the pull of peristaltic activity upon the balloon results in a pressure necrosis of the mucosa in contact with the tube. Berger and Aebi report a case of perforation of the small bowel by the metal tip of the Miller Abbott tube.

Schlucke, Bergen and Dixon report a case of obstruction of the lower ileum due to the coiling of an intestinal tube. Klemminger reported the development of an abscess in matted loops of bowel as a result of a Miller Abbott tube which could not be withdrawn from the small bowel.

Warren and Cattell report a rather unusual complication of intubation. In this case intussusception of the small bowel occurred over a



FIG. 348. Note the leakage of mercury as a result of the rupture of the balloon. Mercury is innocuous in the gastrointestinal tract.

Miller Abbott tube. The balloon acted as a bolus intruding the intussusception. Brunn and Levin report a similar case. In such cases the intussusception is not due to attempts at withdrawal of the tube but is the result of the air-filled balloon behaving like a space-occupying lesion. This is apparent from the fact that the intussusception passes distally into the intussusceptions and not in a retrograde fashion as would occur if the withdrawal attempts caused the intussusception.

Linnbom reported a case of inflammation of the appendix caused by metallic mercury which had leaked out of a Miller Abbott tube. The patient was admitted to the hospital with an obstructive urgency of the rectosigmoid. A Miller Abbott tube was introduced a few days before surgery and mercury was then injected into the balloon. The balloon ruptured and mercury escaped. In 24 hours a fairly large amount of mercury was found in the appendix. When colostomy was performed the appendix was found to be



FIG. 349. Notice the fine dispersion of mercury throughout the colon visualizing it as effectively as a barium enema.



FIG. 350. A catheter passed through the fistulous tract and barium injected to visualize the point of origin of the fistula. Notice the visualization of the left colon. Note also that the mercury present throughout the colon has entirely disappeared.

acutely inflamed and was therefore removed. Gross examination of the appendix showed globules of mercury in its lumen. Microscopic examination verified the diagnosis of acute appendicitis. Chodoff reported the escape of metallic mercury into the intestinal tract after right hemicolectomy. Three intestinal fistulas developed proximal to the anastomosis. At autopsy, the fistulas presented abscesses containing metallic mercury. Hoffman reported the presence within an appendix removed at operation of globules of metallic mercury which had produced no inflammatory change at all. Lindenmuth reported a persistent fecal fistula caused by leakage of mercury from an intestinal tube. In this case, healing of the fistula did not occur until the tract and all the mercury in its wall had been excised. It seemed that the mercury acted as a foreign body preventing the spontaneous closure of the fistulous tract. Our experience with the use of the mercury-tipped metal We believe that in the complications re-

ferred to, one other causative factor was involved. In 1911 Cantor reported an unusual case in which mercury had leaked out of a long tube in such quantity that the globules of the mercury outlined the colon. This case confirmed the observations of Dujardin Beaumetz that the mercury becomes finely divided in the gastrointestinal tract and does not move en masse. In addition, the fistulous tracts present in the patient following an injury to the colon were shown to have globules of mercury in them since mercury was excreted through the fistulous openings. Defunctionizing colostomy resulted in a rapid closure of these tracts. It is thus apparent that fistulous tracts do not necessarily remain persistent because of the presence of metallic mercury within the bowel or within the fistulous tract *per se*. For this to happen, the mercury must become trapped within the tract and act there as a foreign body. This is suggested by the report of Crikelair and Hiratzka

who reported the development of a granulomatous mass containing mercury. Histologic sections of the tissue fragments of the granuloma disclosed numerous small secondary abscesses in the granulation tissue and extensive fibrosis externally. The areas of granuloma reaction disclosed well developed epithelioid tubercles containing foci of active suppuration and in addition tiny globules of mercury were recognizable.

THE USE OF MERCURY IN THE TREATMENT OF INTESTINAL OBSTRUCTION

The use of mercury in the treatment of intestinal obstruction dates back to antiquity. Rhazes (865-923 A.D.) an Arabian physician of such prominence that he was called the Galen of his time pointed out that metallic mercury given by mouth was not toxic. In recording his experiments with this heavy metal he said:

As to pure mercury I believe it not to be very pernicious: it gives rise to abdominal and intestinal pain but afterwards leaves the body as it entered it especially if the subject takes exercise. I gave it to a monkey that I had at my house and things passed as I have said.

Here then we have one of the earliest demonstrations that metallic mercury is innocuous when given by mouth. Many articles were written in the following thousand years describing the oral use of metallic mercury in the treatment of intestinal obstruction.

That metallic mercury enjoyed great popularity between 1780 and 1850 is indicated by the fact that a review of the Index of the Surgeon General for these years reveals more than 50 articles describing the action and use of metallic mercury in the management of intestinal obstruction. One of the most interesting papers on this subject was written by Madden in the Philosophical Transactions in London in 1732. The communication was as follows:

I was present with Dr. Robinson and Mr. Nichols our Surgeon General at the opening of the Body of a Gentleman of Note in this town, who for several years had found great difficulty in going to Stool. This Disorder increased upon him toward the latter End of his Life and he was seized with a Violent Distemper of which I can give you no Description having never attended him.

In order to procure a Passage downward (which I suppose was a principal Complaint) he took by the Advice of a Physician since dead, several Ounces of crude

Mercury at different times without any Relief and at length died.

Upon opening the Abdomen which was very much distended there burst forth a great Quantity of Wind though the Cuts and Stomach were not wounded.

The Stomach was empty and upon opening it we found the inner Coat very much inflamed from one End to the other. We observed in several Places of the small Cuts some scattered grains of crude Mercury and along with them we generally found a black gritty Powder very like Aethiops Mineral which was without doubt the Mercury changed into that Consistence.

This observation by Madden was the first description of what one might find within the gastro-intestinal tract in any patient having taken metallic mercury by mouth.

In 1879 in his doctoral thesis Auguste Mathéon concluded on the basis of his studies that metallic mercury given for intestinal obstruction in dose of 100 to 200 grams was perfectly inert and was the treatment of choice. He believed that metallic mercury was effective in the management of obstruction because of its weight and particularly because of its ability to break up fecal impaction.

As proof of the innocuousness of metallic mercury Mathéon reported that of all the cases he studied in which mercury was given in large doses there was not a single case in which there was the slightest sign of mercury intoxication. He reported a patient in whom the mercury had been present for 22 days asymptotically—in indication of the length of time that mercury could remain in the gastro-intestinal tract without producing intoxication. In addition he noted that in many cases the mercury was expelled partly as a metal and partly as a black powder. The composition of this black powder he believed to be a combination of mercury compounds formed within the bowel by the action of intestinal gases upon the metallic mercury.

The use of mercury in the treatment of intestinal obstruction gradually fell into disrepute so that by 1886 the use of metallic mercury was strongly condemned by many of the leading surgeons of France. Duyardin Barometz in discussing the use of metallic mercury by mouth in the treatment of intestinal obstruction wrote:

"Medical writers of a former day such as Ambroise Pare, Riolan and Belluc have recommended its use in which metallic mercury overcame the intestinal

tacle. A pound or even a pound and a half of this metal would be given in one dose. The patient was then put in a bath and while there two vigorous assistants kept striking him in order to make the mercury fall into the intestine and thus cause the obstruction to disappear. Gentlemen this is a measure that I should be mentioned only to be condemned. Haemus has shown by careful experiments that mercury when introduced in large quantities does not descend in bulk into the large intestine but penetrates slowly globule after globule and this is so true that if the obstacle be removed you do not see the mercury voided in great masses but little by little in particles or globules in the stools and for weeks afterwards this metal is found in the ejecta. Moreover the horizontal position maintained by the patient is a hindrance to the direct action of the mercury on the obstruction. So despite the favorable facts reported by Tessier, Tessier, Houslebeine, Feille, Foucault and Matignon I am of the opinion that mercury should be utterly disregarded in the treatment of intestinal obstruction.

Although mercury is no longer used within the bowel as a method of treating intestinal obstruction its use within the balloon of the long tube has made it possible to greatly simplify such tubes. In addition the percentage of unsuccessful intubations has fallen sharply.

The role of mercury in the balloon of the long intestinal decompression tube appears to be misunderstood. Many surgeons labor under the erroneous belief that it is the weight of the mercury which carries the intestinal tube down the gastrointestinal tract. The effect of gravity upon the weighted head is thought to constitute the propulsive mechanism. This mechanism is utilized in the single lumen Harris tube and in the Miller, Abbott or Johnston tubes.

But if it is weight alone that is desired why not use lead which is heavier than mercury? In addition to being heavy lead is relatively nontoxic in the gastrointestinal tract because it would be excreted before much absorption could occur. However it is quite apparent that although lead is heavier than mercury its other physical properties (*i.e.* being a solid and inert mass) do not fit it for use in an intestinal decompression tube which is expected to pass through narrow passageways with sphincters such as are found in the gastrointestinal tract. Mercury because of its fluidity and cohesive power is very suitable for use in the balloon of a long tube because it literally flows

downward through narrow portions of the bowel and passes sphincters.

Since mercury is to be used in the head end of the intestinal decompression tube such tubes should be constructed to utilize *all* the physical properties of the mercury and not merely its weight. To utilize all the physical properties of the mercury to their best advantage it is necessary to place the mercury in a bag which will give it a free range of motion. We must not limit the free play of the mercury in the balloon if we are to utilize its most desirable properties—fluidity, marked motility and cohesive power. For this reason the Cantor tube was designed to permit the maximum utilization of all the physical properties of metallic mercury and not merely the effect of gravity upon its weighted mass. It should be noted that the ample balloon at the tip of the tube permits a free flow of the metallic mercury. With this mercury trapped in a balloon which permits it to flow freely it is only necessary to place the patient into a position so that the mercury will always have an opportunity to flow downhill or from side to side in order to secure rapid passage of the tube down the gastrointestinal tract. It must be remembered that mercury will not run uphill unless forced upward by peristaltic activity.

Metallic mercury remains liquid under a wide range of temperatures from minus 39 to plus 360 degrees. It is a coherent mobile liquid which does not wet glass or objects placed in it. It remains unchanged in dry air, oxygen, nitrous oxide and carbon dioxide but in damp air it slowly becomes coated with a film of mercurous oxide. It is unaffected by dilute sulfuric acid and even concentrated hydrochloric acid has only slight effect upon it. Its complete insolubility in acids makes mercury an excellent element for use in the head of an intestinal tube because it is completely nontoxic in its metallic form in the gastrointestinal tract. Mercury is quite volatile. A high grade exposure to metallic mercury vapor is much more toxic than when metallic mercury is taken by mouth or through the skin. It is believed that quantities of mercury in the neighborhood of 2 μ g per cubic meter of air when inhaled may cause chronic mercury poisoning. This is characterized by stom-

titis and renal irritation with additional nervous and nutritional disturbances

BALLOONS OF LONG INTESTINAL DECOMPRESSION TUBES TRAPPED IN THE GASTRO INTESTINAL TRACT

One of the rarer complications associated with intestinal intubation is the inability of the intubator to remove the intestinal tube after its use is no longer required. With one type of tube Harris reported a 3 per cent incidence for this unforeseen complication. The reports indicate that this accident has occurred at one time or another with all the tubes which are characterized by the presence of a balloon along the shaft. Although this accident has not happened with the use of the Cantor tube an accident of a similar nature has. This was the loss of an air and mercury filled balloon which had been improperly tied off.

The cause, effect and treatment of this complication have not been the subject of much study. That there is need for elucidation of this complication is evident since several fatalities have been reported as a result of the surgeon's inability to withdraw the tube or his lack of knowledge as to how to cope with the situation.

Cause

The studies of Cantor, Phelps and Felsing upon the effects of the various gases found in the gastro intestinal tract of patients with intestinal obstruction have led to an understanding of the cause of this accident. The studies have demonstrated that all balloons are permeable to all the intestinal gases. Some types of intestinal tube balloons such as those made of latex are five times as permeable to various intestinal gases as those made of Neoprene rubber.

In a review of the case in which the intubator was unable to remove the intestinal decompression tube, one fact was common to all that was the presence of air or gas within the balloons of the tubes in an amount far greater than should be present. In the double lumen tubes it is normally possible to withdraw the air from the balloon thus collapsing it and making its withdrawal simple. Unfortunately, at times knotting of the double lumen tube or obstruction of the inflatable lumen



FIG. 351. Notice the mercury filled balloon of a Cantor tube which had been lost in the gastro intestinal tract. This balloon was excreted within two days.

occurs so that the air which was injected into the balloon remains trapped.

Since nitrogen constitutes 80 per cent of the air trapped within the balloon of these double lumen tubes and since nitrogen diffuses very slowly through the wall of the balloon the consequence to the patient may be serious. This is especially true in the use of the Harris tube whose balloon can contain up to 125 cc of air. Gas distended balloons may obstruct the bowel, cause a tear in the bowel or cause disruption of the anastomosis if such gas filled balloons are pulled through anastomotic areas.

Effect

The effect of a distended gas filled balloon within the gastro intestinal tract depends upon the type of case being intubated. If the patient has been intubated for any type of intestinal distention which is not the result of mechanical obstruction there may be no ill effect whatsoever. The caliber of the bowel is such that in the majority of

in the cases gas filled balloons even when attached to the tube can be passed per rectum if released. This is especially likely to occur when the gastrointestinal tract has been decompressed for in this event there would be an outward diffusion of gas from within the balloon into the decompressed bowel so that when the tube was ultimately passed per rectum the balloon would contain very little gas. If the bowel is not decompressed then a much longer period of time would be required before any of the gas from within the balloon would diffuse into the bowel. In this event a second tube can be passed in order to secure the decompression of the bowel which would then make it possible for the balloon to deflate by the mechanism of diffusion of gases.

In any case in which there is a mechanical obstruction it may be impossible for the gas filled balloons to pass the narrowed area. Once the obstruction has been released the balloons readily pass through and are excreted per rectum. The distended balloons may cause a partially obstructed bowel to become completely obstructed. This is most likely to occur if the balloon is larger than $2\frac{1}{2}$ inches.

If the balloon has been lost within the gastrointestinal tract of a patient with a partial obstruction on a mechanical basis a ball valve type of obstruction might occur. Gas and some liquid stool can frequently pass around the distended balloon through the partially obstructed area. This is apt to give one a false sense of security. The most serious effect of gas trapped or lost balloons occurs in patients who have had bowel resections or bowel surgery of any type. In such cases if it is not recognized that the intubator's inability to remove the tube is due to a distended balloon and if the balloon is distal to the anastomotic line the process of withdrawing the tube may tear the anastomosis.

Treatment

No one treatment is applicable to all cases. The majority of patients in whom there is no decrease in the caliber of the bowel can easily be cured by cutting the tube off at the nose and permitting it to pass per rectum. This it will always do provided there is no obstruction of the bowel distal to the

tube. In the event that the mercury filled balloon of the Cantor tube has been lost the treatment will depend upon whether the bowel has been previously decompressed. If the bowel has been decompressed such balloons will generally pass per rectum the shortest period of time recorded for this to occur being a day and the longest 27 days. In no case was there any injury to the bowel nor were there any untoward effects to the patient during this period of time.

If the bowel has not been decompressed a second tube may be passed in order to obtain satisfactory decompression. The lost balloon will then pass if the bowel has not had its lumen narrowed. If the balloons fail to pass per rectum it is safe to presume that a partial bowel obstruction exists.

An intestinal decompression tube should never be removed from any patient on whom bowel surgery has been performed until a survey film of the abdomen has been taken. This film will localize the position of the tube with reference to the anastomosis. If the balloon is distal to the anastomosis permit it to pass per rectum. If it is proximal to the anastomosis the tube should be removed from above. Decompression of the bowel circumferential to the distended balloon usually helps to deflate the balloon. An occasional case may require operative intervention. If this is done compression of the distended bowel containing the gas filled balloon will rupture the balloon so that it can be removed. To obtain this compression the surgeon need merely grasp the distended bowel between his palms and squeeze upon it.

ERRORS AND SAFEGUARDS IN THE USE OF LONG INTESTINAL DECOMPRESSION TUBES

The effectiveness of the intestinal decompression tube is in direct proportion to the ability of the surgeon who uses it. Above all the proper utilization of the long intestinal decompression tube requires common sense on the part of the intubator. Intubation should not be relegated to the junior intern or nurse on the floor. The attending surgeon must either supervise the passage or pass the tube himself and he must see the patient at frequent intervals during the first 24 hour period. He must determine whether intubation alone is

suitable or whether the patient should be intubated and then operated upon

If the intubator is acquainted with the potentials and characteristics of each type of tube he can avoid many of the errors which have occurred from time to time in their use. Most of these errors are the result of thoughtlessness on the part of the intubator although some may be the result of a lack of knowledge or a lack of care.

When the surgeon orders a long intestinal decompression tube he should specify the type of tube desired. Since the various types of tubes in use today are based upon different propulsive principles they require different techniques for passage. When the surgeon specifies a Miller Abbott tube he should be able to recognize this tube and not accept any other. Similarly if a Cantor tube is asked for an air propulsion tube should not be accepted.

Once he has selected the proper tube the surgeon should examine it to be certain that it is not defective and that its various parts are properly attached. The following list includes some of the things the surgeon should watch for:

1. The metal indicators of the Miller Abbott tube may be reversed. As a result mercury may be injected into the gastrointestinal tract instead of into the balloon. Considerable air may be injected into the bowel in an effort to inflate the balloon if the inflating channel is in the suction channel. Irrigation of the Miller Abbott tube with improperly applied indicators may result in the balloon being filled with solution.
2. Holes in the balloon make proper inflation of the Miller Abbott balloon impossible.
3. Applying the balloon over too short a length of tubing causes angulation of the tube.
4. Ties applied too tightly to the proximal portion of the balloon obstruct the tube.
5. In using the Cantor tube a 21 or 22 gauge needle must be used to insert the mercury and aspirate the air. A larger caliber needle will permit the mercury to leak out and a smaller caliber needle produces a hole so small that it will not function as an effective safety valve.

Errors are also made in the insertion of the intestinal tube. Do not try to push a large metal tipped tube like the Johnston tube through a narrow nose. If this type of tube is to be used pass a small catheter through the nose first and then fasten it to the end of the tube and pull it backward out through the nose. The metal tipped portion is then swallowed and permitted to pass in the usual fashion. In using a Cantor tube remember that you are relying upon a free flow of a highly labile heavy metal. Hyperextend the neck of the patient in inserting the tube so that the nasal passage runs downhill. Do not push the tube uphill into the nose. Do not attempt to intubate a comatose patient.

With the tube in the stomach be certain that there is not too much tubing passed at any one time. Too much tubing causes coiling in the stomach and predisposes to knot formation. The Miller Abbott, Johnston and Harris tubes are calibrated in centimeters. Remember that is usually

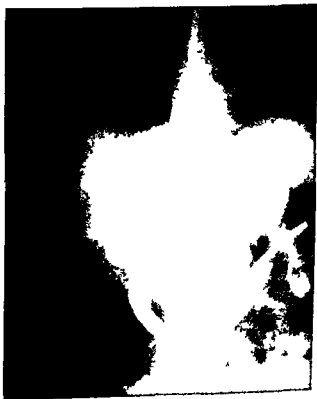


FIG. 352. The effect of leakage of mercury as demonstrated by the fact that only 2 cc. of mercury remained within the tube. As a result the downward passage of the tube is greatly delayed, producing looping of the tube in the duodenum.

requires 60 cm to carry the tube well into the stomach. In the Cantor tube the markings are *S* for stomach, *P* for pylorus, and *D* for duodenum. When the directions call for *S* to be at the nose do not insert the tube to the *D* mark. Proper intubation will result in better decompression.

Failure to keep the tubes well lubricated and the mucosa of the nasopharynx moist may result in the development of otitis media due to irritation of the ostium of the Eustachian tube. In addition try to keep the tube away from these ostia.

The intestinal decompression tube may become plugged with intestinal contents as noted by Johnston who reported a plugging of the tube by orange pulp. Hinson reported a valve type of obstruction of the tube due to a blood clot in the lumen of the tube. This permitted fluid to be injected into the bowel from above but would not permit aspiration of the bowel contents. Such accidents are likely to occur in tubes with a small lumen.

If the patient being intubated has been subjected to a gastrectomy or a gastro-enterostomy a change in the technic of intubation is necessary. This is due to the change in the anatomic position of the gastric outlet as well as the anatomic position of the proximal and distal jejunal loops. Remember that after gastrectomy and gastro-enterostomy the gastric stoma is at the greater curvature. This being the case in intubating these patients standing the patient up and ambulation would carry the mobile heavy tube head directly to the outlet of the stomach on the greater curvature because such positions cause the greater curvature to become the most dependent part of the stomach. In addition it is helpful to know just which type of anastomosis was performed. If the proximal loop is anastomosed to the lesser curvature and the distal loop to the greater curvature ambulation will rapidly result in a successful intubation. If however the proximal loop is anastomosed to the greater curvature and the distal loop to the lesser curvature ambulation may cause the mercury filled tube head to become trapped in the proximal loop. In such cases withdrawal of the tube into the stomach and turning the patient on the right side will bring the stomach outlet to a downhill position. As a result intubation can be performed successfully.

In using an air filled balloon tipped tube remember that an excessively inflated balloon may cause obstruction by itself. Once the intestinal distention has subsided following successful intubation a reappearance of the distention with the suction active should create a suspicion that balloon blockage has occurred. Deflate the balloon to note whether the obstructive signs disappear. If they do the diagnosis is obvious. If the obstructive signs do not disappear surgery may be indicated.

SWALLOWED LONG INTESTINAL DECOMPRESSION TUBES

There are several ways in which this may occur. The patient may deliberately swallow the intestinal tube. The intern or nurse may permit the intestinal tube to be swallowed as a means of removal. It may be difficult or impossible to remove the tube from the nose and hence the best method of removal would be to allow it to pass downward for excretion per rectum. If the intestinal decompression tube has free peristaltic activity may be sufficiently forceful so that the tube emerges per rectum.

The patient may deliberately swallow the intestinal decompression tube to satisfy some mental aberration. Such patients are in the same group as those who swallow coins, pins, hairpins and a wide variety of other foreign bodies. In such cases if there is no obstruction to the intestinal stream the tube will invariably pass through and be excreted per anus. The length of time that this takes varies depending upon peristaltic activity and tonus of the bowel as well as the presence or absence of kinks or angulations in the bowel from adhesions which may cause partial obstruction. In this event the downward passage of the intestinal tube would be blocked at the level of the partial obstruction. The failure of the tube to be excreted in a reasonable period of time need not necessitate surgical intervention in the absence of signs of bowel obstruction. As short a time as four hours and as long a time as 18 days may elapse between the time the tube was swallowed and the time it is excreted per rectum without harming the patient. On occasion such tubes may fail to pass as a result of adhesions which so kink and angulate the bowel that the tube cannot pass through this narrowed segment of bowel. Failure of an in

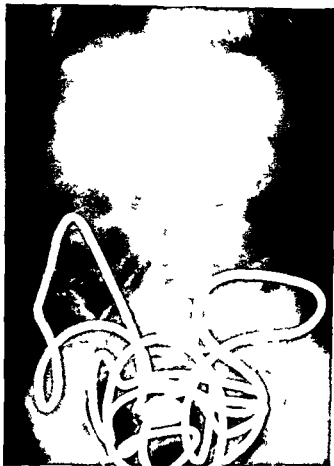


FIG. 33 Intestinal decompression tube swallowed by the patient and lost into the colon. The tube was excreted in approximately three hours following an enema.

testinal decompression tube to pass per rectum when associated with an increasing or recurring intestinal distention is suggestive of a partial intestinal obstruction being present distal to the tube. In this event the treatment is surgical. Relieving the obstructing process distal to the tube will invariably result in an easy passage of the tube through the previously obstructed area. When the obstructing process is due to adhesions (which account for most of such cases) simple lysis of the adhesive bands is sufficient. Enterotomy to remove the tube is not required and needlessly exposes the patient to peritoneal soiling. The tube head may easily be milked into the cecum from which it is readily passed per rectum.

The internurcic or resident may permit the intestinal tube to be swallowed by the patient as a means of removal. This is particularly apt to occur when an unstimulated nurcic or internurcic

is to remove a tube which has been passed far down the gastro intestinal tract. On occasion the nurcic may consider it easier for the patient to pass the tube per rectum rather than have it removed from above.

Two widely different conditions may make it difficult or impossible to remove the tube from above. The first condition is the result of an unrelenting sphincteric spasm in a nervous, highly apprehensive individual. The second is the result of permeation of intestinal gases within the balloon of the intestinal tube. In this latter event the balloon becomes so large that its removal from above is very difficult or even impossible.

In cases in which it is difficult to remove the tube from above the tube may be deliberately permitted to pass downward for excretion per rectum. The nervous, apprehensive high strung individual may become so disturbed when one attempts to remove the intestinal tube from above that severe and persistent sphincteric spasm occurs which effectively trap the tube within the bowel. In such cases giving sodium luminal and atropine by injection relieves the patient so that removal from above may be made possible. On one occasion despite this procedure it was impossible to remove the tube from above. The tube was then cut off at the nose and permitted to be excreted per rectum.

The problem of a balloon so filled with intestinal gases that the bulk of the tube head makes removal from above difficult if not impossible is most apt to occur with the Harris tube because the balloon of this tube is 6 inches long and may take up as much as 125 cc of gas. Harris noted this occurrence and advised permitting the tube to be excreted per rectum. This usually occurred in the absence of bowel obstruction distal to the tube head. If the bowel distal to the tube head is kinked, angulated, narrowed or the site of an intestinal anastomosis it may be necessary to operate upon the patient before the tube can be removed. In the absence of signs or symptoms of bowel obstruction this should not be done. Convulsions and watchful waiting will often be rewarded by the ultimate passage of the tube. The development of signs of bowel obstruction in such cases is an indi-



FIG. 354 Intestinal decompression tube emerging from the rectum. When this occurs the use of the tube is no longer required and it should therefore be removed from below.

cation for immediate surgery. In this case simply squeezing the distended bowel between the palms of the hands causes the balloon to break. This permits the tube to be simply removed via a minimal enterotomy or if the obstructing process has been released such tubes will readily be excreted per rectum. In no event should the tube be removed with an inflated balloon. To do so would necessitate an unnecessarily long enterotomy incision. If the tube had not been swallowed by the patient it could have been readily removed from above after the collapse of the balloon.

There have been case reports of intussusception of the jejunum or ileum as a result of the presence of an inflated Miller Abbott tube. In such cases the inflated balloon acts much like a space-occupying tumor and furnishes the nidus for the intussusception. Removal of the tube from above may then be impossible. In any event surgical correction of the intussusception is imperative in such cases.

In some patients the peristaltic activity may be so active that the tube head may be found to have emerged per rectum. In cases of this type the need for intubation no longer exists. For esthetic reasons such tubes are released at the nose and pulled out per rectum.

PARENTERAL FLUIDS AND INTESTINAL OBSTRUCTION

ROBERT F. I. BERRY, M.D.

The sound treatment of the majority of intestinal obstructions is closely integrated with the intelligent use of parenteral fluids. Although our knowledge of this field has been implemented by a great deal of experimental and clinical investigation during the past decades, even the most skillful use lacks perfection. Parenteral fluids remain an incomplete but reasonable satisfactory substitutive method of providing many nutritional elements when the gastrointestinal tract has been temporarily decommissioned by intestinal obstruction.

The rapidly accumulating literature on parenteral fluids has given rise to a certain degree of confusion which is the inevitable result of incomplete knowledge of many phases of related body physiology, emphasis on the chemical anatomy of body fluids, and use of complex terminology to describe salt concentrations and movements of body fluids.

This chapter will attempt to present a practical approach which has been found to be workable in clinical experience. It is important to point out at the onset that blood and urine chemistries, as determined in the laboratory, are not fixed points about which fluid balance revolves as a clinical problem. Laboratory data often provide important adjunctive information but the important considerations are the patient's history and what the clinician sees, feels, and hears.

The approach to this subject will be threefold: first a short review of present concepts of water and electrolyte metabolism; second the abnormal

metabolic state produced by intestinal obstruction; and third the use of parenteral fluids.

WATER METABOLISM

The amounts of indigenous salts dissolved in the water of the body are jealously guarded constant in health. Gastrointestinal fluids lost during intestinal obstruction contain both water and salt. It is essential, however, to discuss separately the metabolism of body water and salt because of physiologic differences related to loss and excretion. Since the need for water and salt is never exactly the same, they must be thought of as two separate entities.

Amount and Distribution of Body Water

Except in the case of the very obese, the major part of body weight (B_w)^{*} is water. With the exception of bone, fat-free tissues are about 70 per cent water. However, it is incorrect to state that 70 per cent of the body is water, since fat, which contains only 10 per cent water, may constitute an appreciable percentage of B_w . The percentage of B_w as water varies inversely with the content of fat tissue. Determinations with deuterium oxide indicate that the majority of individuals have from 0 to 65 per cent of B_w as water. According to Schloerb and his co-workers, in the very obese this figure may fall as low as

The symbol B_w will subsequently be used to designate body weight.

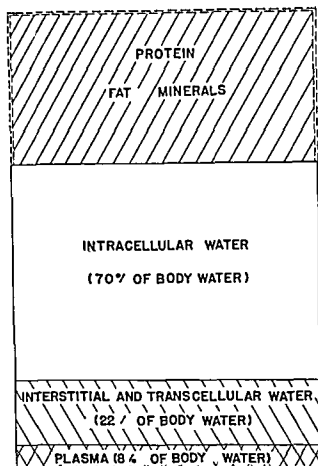


FIG 355 Composition of the human body. The per centage of B_w as water varies principally with the fat content but is usually 50-65 per cent of the B_w . The remainder is made up of proteins, fat, and minerals. With relative greater constancy about 10 per cent of total body water is in the cells while the remainder constitutes the water of extracellular fluid.

35 per cent while the content in infants and very thin adults may exceed 70 per cent.

Although the percentage of B_w as water has in individual variability the percentage of total body water found inside the cells (intracellular water) and outside the cells (extracellular water) remains fairly constant. Hardy and Drabkin have demonstrated that extracellular water has greater proportionality to total body water than B_w . About 70 per cent of total body water is intracellular in position while the remaining 30 per cent is in the extracellular phase (Figure 355). Approximately 25 per cent of extracellular water (4 to 5 per cent B_w) is intravascular (plasma). The remaining 75

per cent is extravascular constituting an interphase between water in blood vessels and that within cells (interstitial water) or present as spinal fluid, gastric juice, etc. (transcellular water). For practical considerations transcellular fluids are considered part of the interstitial fluid compartment.

Gain and Loss of Water

In health water is principally provided by oral intake of food and drink. An insensible gain of water is further contributed by two types of cellular metabolic action.

1 The release of intracellular water necessary to keep carbohydrate and protein stores in solution during periods that such stores are not adequately repleted (water of solution).

2 Water resulting from the catabolizing of nutritional elements for energy needs (water of oxidation).

The average gain of insensible water is about 0.04 per cent of B_w per hour in the resting state with nothing being taken by mouth. For a 70-kg non-fasting man this would amount to 336 ml (or cc) in a 24-hour period. The minimal average and maximal gains of water through the intestine and by the metabolic water of solution and oxidation are shown in Table 2.

Insensible gain can be only a rough estimate under ordinary clinical conditions and has diminishing importance during such disease states as

TABLE 2

The average sensill and insensible gain of water per 24-hour period and the minimal and maximal rates of water gain expressed as percentage of body weight per hour

	$\frac{1}{100}$ ge (ml / 4 h g)	$\frac{1}{100}$ ml Ma mal (B per ho)
Sensible gain by eating and drinking	1275-2275	0.00 3.9 (diabetes insipidus)
Insensible gain from water of solution and oxidation	336 (non fasting) 70 kg male	0.01 0.08

Data from MOYER, C. A. *Fluid Balance, A Clinical Manual*. Chicago, Illinois: The Year Book Publishers, Inc. 1952. and ADOLPH, F. F. *Physiological Regulations*. Lancaster, Pa.: Jaques Cattell Press, 1943.

intestinal obstruction when large turnovers of body water may take place. Water is normally lost by

- 1 Vaporization from the lungs and skin
- 2 Sweating
- 3 Fecal discharge
- 4 Urine output

Water vapor formed within the body is dissipated through the lungs and skin. Activity of the sweat glands is not necessary for loss from the body surface. The average 24 hour loss is about 1000 ml (Table 3).

Loss of water by sweating has great variability. A resting man begins to sweat at an environmental temperature of 78 degrees Fahrenheit in a relative humidity of 60 per cent and at 86 degrees if the relative humidity is 30 per cent. As much as 3500 ml per hour can be lost as sweat (Table 2). Sweat differs from insensible loss in that it contains variable concentrations of sodium salts, the maximum being about 90 mEq per liter. Loss of water by vaporization and sweating is obligatory and continues even though a deficit of total body water exists.

Fecal loss of water is usually less than 200 ml per day. It is possible, however, to lose as much as

4 per cent of B_w per hour during severe diarrhea. In just a few hours this type of loss could be fatal due to circulatory failure secondary to acute extracellular fluid depletion.

The lungs, skin and intestine do not regulate the water and salt composition of body fluid, however. This is a function of the intact kidney. The volume and composition of the urine are variable depending upon the demand being made on renal function in order to maintain the normal constancy of body fluids.

Renal Excretion of Water

The kidneys excrete daily 35 to 40 gm of solids that are chiefly waste products of nitrogenous metabolism. A minimum of 15 ml of water is required to dissolve 1 gm of these waste products. Because of the limited concentration power of the human kidney, an irreducible amount of water is necessary for adequate excretion of nitrogenous wastes. Lashmet and Newberg have demonstrated that at urine specific gravity of 1.035 \pm .00 ml of urine are required to prevent accumulation of nitrogenous wastes. Diminishing concentration power requires greater volume. At specific gravity of 1.010, 1500 ml of urine are necessary for adequate waste elimination.

According to Homer Smith, the ability of the healthy kidney to chlorate a concentrated urine is dependent upon water absorption from the filtrate delivered to the distal tubule and has been chiefly related to neurohumoral hypothalamic-pituitary mechanisms that result in release of the antidiuretic hormone (ADH) from the neurohypophysis. Verney has postulated that the chloration of this hormone depends upon osmoreceptors located in the hypothalamus that are extremely sensitive to the smallest change in the concentration relationship.

A specific gravity of 1.035 approaches the maximum observed in the human. Urine specific gravity depends upon the weight of dissolved solids compared to distilled water. It is but a rough estimate of the renal tubular concentrating ability. A more accurate method is the determination of the osmotic activity of the urine by measuring the freezing point depression. The amount of depression reflects the number of osmotically active particles present. Such determinations are impractical for clinical use, however, and within limits the specific gravity can be used as a clinical guide for the renal concentrating ability.

TABLE 3

The average sensible and insensible loss of water per 4 hour period and the minimal and maximal rates of water loss expressed as percent age of body weight per hour

	Average (ml / 4 h s.)	Minimal (% Bw per hour)	Maximal (% Bw per hour)
Kidney (sensible loss)	800-1500	0.03	2.0
Intestine (sensible loss)	125	0.00	4.0
			(chole- cystostomy, diarrhea)
Lungs and skin (insensible loss)	600-800	0.03	0.12
		(normal in water)	(fever and activity)
Skin (insensible loss by sweating)	none	0.00	5.0
			(sweating)

Data from Moyer, C. A. *Fluid Balance*. J. Clin. Invest. Chicago, Illinois: The Year Book Publishers, Inc., 1952 and Aronow, L. J. *Physiological Regulation*. Lancaster, Pa.: James Cattell Press, 1943.

tion hip between extracellular water and electrolyte. The intake of water in excess of metabolic needs is followed by a depression in the elaboration of ADH, diuresis and maintenance of extracellular osmolarity. Conversely, hyposmolarity induced by inadequate intake of water is followed by increased production of ADH, augmented renal tubular absorption and conservation of body water. Unpleasant stimuli can also produce antidiuresis. This is probably related to the same mechanism. Other antidiuretic substances may exist. Their nature and mode of action await further investigation as do many of the problems of diuresis, antidiuresis.

Factors Controlling the Distribution of Water

Movement across capillary membranes from plasma into interstitial fluid is principally one of passive diffusional osmosis with water passing from areas of greater to those of lesser concentration. This in turn can be modified by renal function within certain physiologic limits, as the intact kidney jealously guards the ratio of sodium salts to water, excreting or absorbing these constituents in accordance with adjustments necessary to maintain normal osmolarity. Moyer has shown this ability may have lessened effectiveness following injury or operation.

The mechanisms of water exchange across tissue cell membranes remain poorly understood. Because of the apparently higher osmotic activity of intracellular fluid, increased attention has been given during the past few years to the physiologic considerations of such differential osmotic gradients. Zuckerman and his co-workers have observed the water content of tissues to change as much as 5 per cent after the administration of adrenal steroid hormones without changes in the extracellular osmotic pressure. Robinson has demonstrated that

† Osmolarity depends upon the number of osmotically active particles present. The salts inherent to extracellular fluid exercise the greatest osmotic activity because of their high degree of ionization. The number of osmotically active particles per unit of extracellular fluid is therefore kept constant by renal excretion or absorption of water depending upon the demand being made on the kidney to maintain the homeostasis of extracellular osmotic pressure.

tissues placed in isotonic solutions swell. This can be negated by hypertonic environment and aggravated by chilling, low pH, anaerobic conditions or cyanide poisoning. The observation has been made that intracellular fluids are normally hypertonic to their environment and that the entrance of water is prevented by the constant active outward transfer of water across the cell membrane. Such pumping action would require energy and the amount of water passing outwardly from cells has been found to be proportional to their oxygen consumption.

Grudino and Levitt have shown that adrenalectomized animals transfer water from the extracellular position into the cell. This may possibly be responsible for signs of water intoxication observed in certain acute adrenal insufficiency states. The administration of adrenal steroids can prevent this phenomenon. The observation that the 11-17 oxygenated steroids may be required to promote the transport of water out of the cells is of great interest but the mechanisms involved have yet to be determined.

It becomes obvious that even in health the movements of body water present phenomena not fully understood. Certainly then the complex aberrations of body water physiology produced or modified by disease, injury or operation are even less well understood and should be approached with full awareness of our lack of knowledge. The application of rigid dogma or theory has little place in the diagnosis and treatment of problems of water balance.

ELECTROLYTE METABOLISM

Electrolytes are defined as substances which when in solution are capable of conducting an electric current by means of their ions. The electrolytes of body fluids are chiefly sodium and potassium salts but also include those of calcium and magnesium. Because of their osmotic activity and essentiality to intracellular metabolic processes, the maintenance of normal qualitative and quantitative relationships of the body fluid electrolytes is of the greatest importance. The term milligrams per cent, previously used to designate the quantities of these substances, has been superseded by

equivalents or milliequivalents (mEq) § This has the advantage of substituting physiologic (osmotic) activity for weight as well as permitting a simple expression for the concentration of each ion. The use of milliequivalents ($\frac{1}{1000}$ of an equivalent) rather than equivalents expedites rapid calculation by avoiding fractions for example 5 mEq of potassium is equal to 0.005 equivalents of the same ion. The formula for the conversion of milligrams per cent (mg %) to milliequivalents (mEq) is as follows:

$$\text{milliequivalent} = \frac{\text{mg \%} \times 10 \text{ (converts mg/100 ml to mg/liter)} \times \text{valence}}{\text{atomic weight}}$$

§ An equivalent of a substance is that amount which will combine with or displace eight parts by weight of oxygen. Weight may have little relationship to physiologic (osmotic) activity. Plasma proteins weighing 69,000 mg per liter have a chemical combining power of only 16 mEq. Sodium however weighing 3260 mg per liter of plasma has a chemical combining power of 142 mEq.

Electrolyte Concentrations

The same electrolytes are present in extracellular and intracellular fluids but have considerable variability in their concentrations. Ionic constituents of the fluid compartments expressed in mEq are shown in Figure 356. About 90 per cent of ions with a positive charge in the extracellular fluid are sodium (142 mEq). The amount of sodium available to extracellular fluid determines its volume and almost all of its osmotic activity.

The principal negatively charged ion in the extracellular fluid is chloride (103 mEq). The primary difference in the chemical make up of plasma and interstitial fluid is the protein concentration. Plasma has 16 mEq of protein as compared to an average of 2 mEq in interstitial fluid. The Gibbs

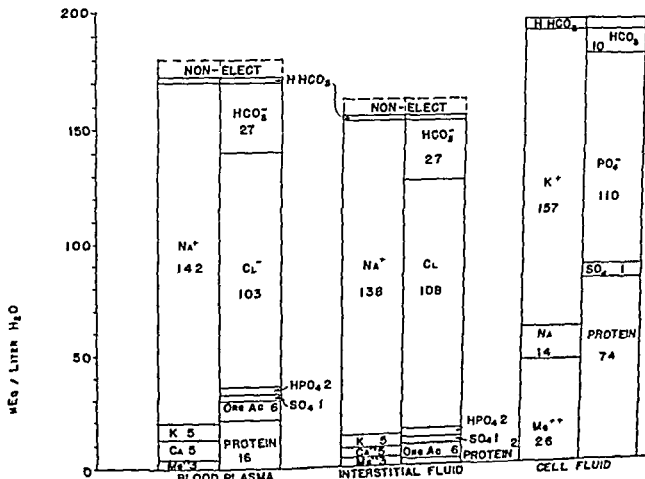


FIG. 356. The electrolyte constituents of plasma, interstitial fluid and cell fluid.

Donnan effect[†] because of this difference in protein content results in a slightly lower sodium concentration (135 ml q) and an equivalent increase in chloride (108 ml q). Only about two thirds of the body sodium is found in the extracellular phase. The remainder is found in the cells of soft tissues and in bone. Ickman and his co-workers have estimated that about 40 per cent of the sodium in bone is exchangeable with that in the extracellular phase. The difference between sodium and chloride concentrations in extracellular fluid may become of considerable importance during fluid therapy as isotonic 0.9 per cent sodium chloride solutions provide about 40 per cent excess chloride ions when compared to the normal concentration in plasma and interstitial fluid.

The quantitative difference in electrolytes of intracellular fluid is quite apparent (Figure 356). The intracellular concentration of sodium given as 14 ml q is not agreed upon by all investigators. It is now recognized that chloride can be present in the cell.

Sodium Metabolism

The normal intake of sodium salts has considerable variability and depends upon the content in food and drink as well as the individual taste for seasoning. The average daily intake is 7 to 10 gm. In the absence of excessive sweating, abnormal loss from the gastrointestinal tract or abnormal sodium retention, all but a very small fraction of ingested sodium is excreted in the urine. The difference between intake and renal output represents that lost by normal sweating and in the feces. A small daily sodium salt intake is not a hazard to health. As a matter of fact, in certain disease states of the cardiovascular system, therapy includes the limitation of sodium intake to less than 1 gm per day for long periods of time.

The normal kidney rigidly conserves sodium during cessation of intake. Urinary output of this electrolyte may approach zero after three to four days of complete deprivation. This important homeostatic mechanism insures minimal renal de-

pletion of the most important electrolyte in extracellular fluid.

The factors influencing the speed of exchange and the ratio of extracellular to intracellular sodium are poorly understood. Cooke and Segar have suggested that this relationship is dependent upon the pH of body fluids rather than being a quantitative effect. It is entirely possible that in intracellular sodium becomes extracellular during acidosis to aid in the defense of extracellular pH. Shifts of an important electrolyte like sodium from its intracellular position are undoubtedly accompanied by cellular metabolic disturbances which have yet to be identified.

The control of the renal excretion of sodium is complex. About 80 to 85 per cent of all fluid filtered through the glomerulus by the normal kidney is absorbed in the proximal tubule. The reabsorption of sodium is an active process necessitating tubular work. This is accompanied by passive back diffusion of water mediated by the necessity of maintaining iso-osmotic pressure. The active reabsorption of sodium is in part influenced by steroids from the adrenal cortex that resemble desoxycorticosterone acetate (DCA) in their action. The inability of the kidney to conserve sodium efficiently in such states of adrenal insufficiency as Addison's disease is well recognized. Only about 2 per cent of the filtered load of sodium is affected by these steroids, however. Absorption of sodium continues in the distal kidney tubule. If the filtrate being delivered to the distal tubule has a low concentration of sodium, almost all of this electrolyte may be absorbed. Higher concentrations of sodium are not completely absorbed and the excess is excreted in the urine. The mechanisms by which sodium is absorbed or excreted in the distal tubule are importantly related to the antagonistic action of the antidiuretic hormone from the neurohypophysis and the secondary effect of adrenal steroids elaborated in response to the adrenocorticotrophic hormone from the adenohypophysis (anterior lobe of the pituitary gland). However, many of the aspects of sodium absorption and excretion by the kidney remain to be determined.

Potassium Metabolism

The vagaries of potassium metabolism are even less well understood than those of sodium. Most

[†] When two solutions are separated by a membrane which is permeable to some of the ions but not all of them, an irregular distribution of the ions is the result. The two solutions therefore differ in osmotic and hydrostatic pressure.

foods have a generous potassium content so that a balanced diet provides this electrolyte far in excess of ordinary needs. There is considerable difference in renal conservation of sodium and potassium however. During acute potassium deprivation the kidney is unable to rigidly conserve this ion. The result is a continued attrition of potassium stores, the replacement of which becomes important. The use of sodium containing parenteral replacement fluids further increases the renal loss of potassium during cessation of oral intake. The serum potassium tends to vary inversely with bicarbonate and an inverse ratio of serum sodium/potassium has been observed during acute sodium salt loss.

In contradistinction to sodium, the extracellular osmotic and volumetric functions of potassium are not important. Manery has stated that the earlier concept of a mol for mol exchange of extracellular sodium for intracellular potassium in states of potassium depletion is no longer tenable. The sodium gain and the potassium loss from the cell which occur on low potassium diets are two separate processes as shown during the recovery phase in experimental animals. It would seem that in vestigations designed to test the idea that sodium and potassium occupy different positions within the cell fulfill different functions and are controlled by different mechanisms should be more productive than those based upon the theories of simple exchange.

The extracellular function of potassium is related to the conduction of neuromuscular mechanisms, particularly those of the heart. In the cell a certain amount of potassium is bound by protein and cannot be released until the death of the cell. The remaining portion of potassium passes freely across the cell membrane. Because of its cellular concentration, potassium is related to many important aspects of intracellular activity, particularly those of carbohydrate metabolism. The exact nature of the role of cell potassium however awaits further investigation.

The mechanisms controlling potassium excretion by the kidney are poorly understood. Apparently all filtered potassium is reabsorbed by the activity of the proximal tubule with the result that the urinary concentration is dependent upon distal tubule excretion. Acetazolamide has been identified with

increased loss of urine potassium. Berliner, Kennedy and Hilton have postulated that under the circumstances potassium is exchanged for sodium in the filtrate and that this mechanism is not so much dependent upon pH as upon the partial pressure of CO_2 (pCO_2). It would appear therefore that under certain circumstances the potassium excretion as well as that of chloride/hydrogen ion bicarbonate and ammonia is important in the renal defense of total body pH and can be modified in conditions in which the pCO_2 is increased.

Chloride Metabolism

The imposing concentration of chloride in extracellular fluid rather than some other ion such as sulfate would seem to be related to the teleologic development of the electrolyte pattern of extracellular fluid in humans. Its apparent functions are to provide anions for maintenance of electric neutrality and to aid in the homeostasis of pH. The renal excretion is closely associated with the defense of the hydrogen ion concentration of extracellular fluid. Moyer has stated that the concentration of chloride only in whole blood is *critical value* in clinical problems of fluid balance.

THE ABNORMAL METABOLIC STATE PRODUCED BY INTESTINAL OBSTRUCTION

Because intestinal obstructions prevent the normal oral intake of food and water, a state of involuntary starvation is invariably attendant. The metabolic defect is compounded and complicated further by the rapid and continued loss of gastro-intestinal contents through vomiting, intubation and immobilization into the lumen and wall of the intestine and peritoneal cavity. Although starvation and the loss of gastro-intestinal fluid are common to all obstructions, a number of other considerations affect the type of metabolic defect produced and the prognosis for recovery.

Site of the Obstruction and Relationship to the Metabolic Defect

Obstructions of the esophagus, whether benign or malignant, produce a relatively low percentage of protein in the digesta depending upon how completely the lumen is occluded. The

result is a progressive depletion of body fat reserves and protein catabolism for energy needs. As the fat stores approach depletion a relatively greater catabolism of protein is necessary in order to meet mandatory energy requirements. The important effects of starvation with depletion of protein reserves have been well recognized.

- 1 Diminished red blood cell mass and contracted plasma volume
- 2 Diminished serum protein concentrations reducing the colloid osmotic pressure of plasma thus enhancing edema formation
- 3 Depletion of proteins of all body tissues and particularly those of important parenchymatous organs as the liver and heart
- 4 Poor tissue healing
- 5 Depressed antibody formation
- 6 Depletion of the hormone and enzyme systems
- 7 A labile cardiovascular system that poorly tolerates anesthetics and operation

The magnitude of the metabolic defect due to

inadequate intake of energy calories and protein in esophageal disease is equalled and may even be exceeded by obstructions at the pylorus of the stomach or by obstructions of the small bowel particularly if increased tissue catabolism is concomitant because of fever and peritonitis. In addition to starvation obstructing lesions of the pylorus and small bowel are attended by serious acute losses of elements of extracellular and intracellular fluids which may impose a grave threat to survival long before considerations of malnutrition become of serious import.

It has been well demonstrated that the constituency of fluids lost by vomiting or tube aspiration is variable depending upon the site of obstruction (Tables 4 and 5 and Figure 357). Pyloric obstructions secondary to active peptic ulcers have a high chloride to sodium ratio in the gastric juice. Obstruction of the pylorus due to carcinoma of the stomach usually prevents a high chloride to sodium ratio but not of the magnitude observed in peptic ulcer disease. Loss of chloride in excess of

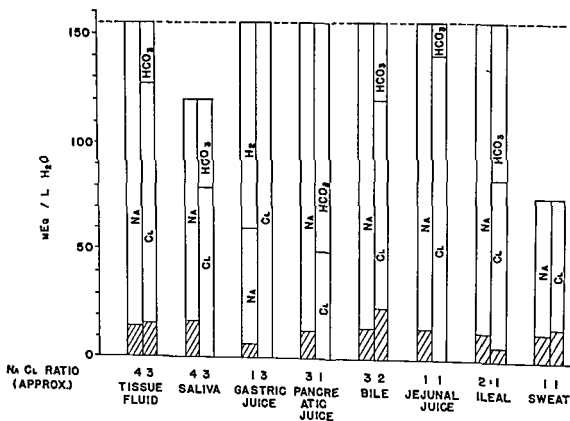


FIG 357 Electrolyte composition of gastro intestinal secretions

TABLE 4

Total volume of digestive secretions produced in 24 hours
in an average adult

Saliva	1 00 ml
Gastric secretion	2500 ml
Bile	500 ml
Pancreatic juice	700 ml
Intestinal secretion	3000 ml

Data from GAMBLE, J. L. *Chemical Anatomy Physiology and Pathology of Extracellular Fluid* Cambridge Mass., Harvard University Press 1947

sodium results initially in renal excretion of sodium as the kidney attempts to maintain a normal chloride to sodium ratio in the plasma. As sodium depletion continues however progressively less sodium and more ammonia is excreted by the kidney because the attempt to maintain normal pH becomes secondary to maintaining an effective volume of extracellular fluid. If uncorrected such losses result in alkalosis, an increased CO content of plasma and a diminished extracellular fluid volume.

Fluid lost in small bowel obstructions tends to have an electrolyte concentration similar to plasma. Large bowel obstructions are usually not attended by serious loss of body fluids unless an incompetent ileocecal valve permits a clinical picture that has relative equivalence to that of small bowel obstruction.

Other Factors Affecting the Metabolic Defect

Duration It is axiomatic that the longer a correctable intestinal obstruction is permitted to go untreated the greater will be the metabolic defect and the more serious the illness. Neglected obstructions not only deplete the body of fat and protein by starvation but the extracellular fluid loss may have reached a critical state and be attended by disturbances of acid base balance. Chronic intestinal obstruction can present a most difficult problem because of inability to maintain adequate nutrition.

Previous Operation The onset of postoperative intestinal obstruction adds a new insult to previous injury. There is some degree of adynamic ileus following any major abdominal operation. Although the majority quickly resolve themselves a few may be most stubborn. Persistent ileus

TABLE 5

Range and averages of sodium potassium and chloride found in intestinal contents *

	Sodium (mEq)	Potassium (mEq)	Chloride (mEq)
Gastric (fasting)	60.4	9.2	84.0
	9-116	0.5-32.5	7.8-154.5
Small bowel	111.3	4.6	104.2
	82.0-147.9	2.3-8.0	43-137
Ileostomy (recent)	129.4	11.2	116.2
	105.4-143.7	5.9-29.3	43-137
Cecostomy	52.5	7.9	42.5

* The great magnitude of range of the electrolytes may necessitate daily determination of long tube aspirate in prolonged and difficult obstructions to determine accurately its ionic constituency.

Data from LOCKWOOD, J. B. and RANDALL, H. T. *Bull. New York Acad. Med.* 1949 25: 228.

should be closely scrutinized to rule out an extracellular fluid deficit or possibly a potassium deficiency as its cause.

Obstructions produced by acute adhesive peritonitis such as those following a ruptured appendix will spontaneously resolve themselves in the great majority of cases without operative interference.

Unfortunately certain mechanical obstructions require a second operation for their correction thus increasing the hazards of malnutrition and potential fluid imbalance states.

General Health Nutrition and Age A healthy, vigorous, relatively young individual has greater tolerance and wider latitude of adaptation to the stress imposed by intestinal obstructions. The incursions of cancer or other debilitating disease upon the general body economy may be of great importance in prognosis. Because most intestinal obstructions represent surgical emergencies, operation is less well tolerated by those in the advanced age groups. Elderly patients have a greater sensitivity to fluid imbalance states and do not adjust as well to acid base disturbances as do younger patients.

Infection Obstructions concomitant with peritonitis or other intra-abdominal inflammatory conditions further complicate the metabolic problems by augmenting the distributional shifts of extracellular fluid and the loss of plasma proteins in addition to increasing the catabolic effect by mobilizing

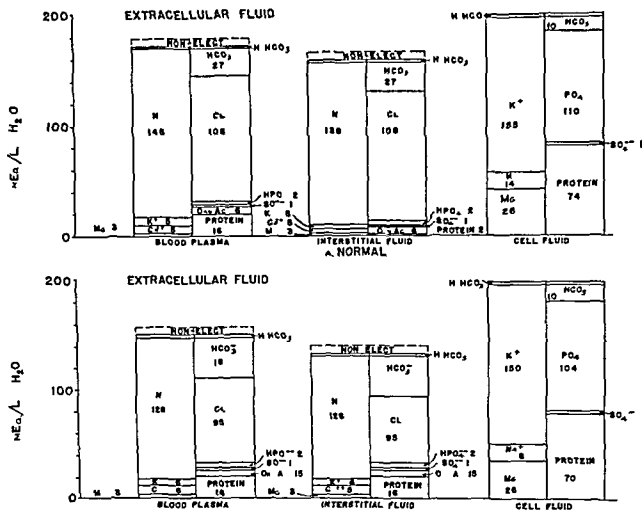


FIG 358 Metabolic acidosis. A loss of intestinal fluid containing a relatively greater amount of sodium than chloride results in lowering of both extracellular sodium and chloride. The relatively greater sodium loss is reflected by a lowering of fixed base bicarbonate from 27 to 18 mEq. Cellular changes are chiefly reflected by a loss of cell sodium as well as lower cell potassium.

tissues. The deleterious effect from the absorption of bacterial products does little to aid a favorable prognosis.

The Effects of Sodium Depletion. In the absence of significant hepatic, renal, or cardiac disease, the two major causes of serious sodium loss are excessive sweating and disease of the gastrointestinal tract. Because of the sodium content of gastrointestinal secretions, the loss of this electrolyte in intestinal obstructions is attended by abnormalities in the volume and sodium concentration of extracellular fluid (Table 5).

Volumetric Depletion of the Extracellular Fluid. The rapid loss of gastrointestinal fluids

containing approximately the same salt constituents as plasma produces parallel depletion of sodium salts and water. This is particularly characteristic of obstructions of the ileum. The most striking picture of this condition is obtained from the physical examination and is, by large odds, the most important method of making the diagnosis. The initial symptoms are referable to a contraction of the plasma and interstitial fluid volumes. Weakness and apathy are usually apparent. The extremities are somewhat cool, and the nail beds may be slightly cyanotic. Peripheral venous filling may be sluggish and stripping of the veins on the dorsum of the hand reveals a diminished rate and vol-

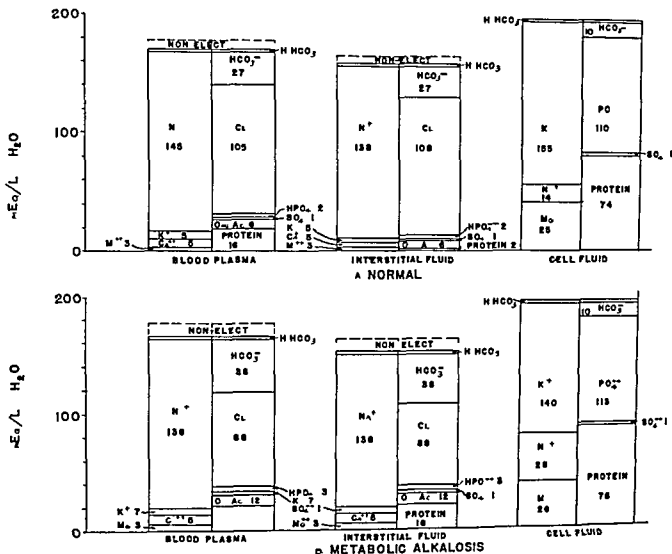


FIG. 339 Metabolic alkalosis. Loss of gastric secretions by vomiting, or aspiration results in a lowering of both sodium and chloride but a relatively greater lowering of chloride. The net result is an increase in the percentage of fixed base bicarbonate. Plasma levels of potassium may be normal, increased or decreased depending upon variables which remain poorly understood. Cellular changes are reflected by an apparent intracellular gain of sodium and loss of potassium. This is not a simple mol for mol exchange but probably represents two different control systems. The movement of potassium into and out of cells is poorly understood.

time of fill. Increasingly greater volumetric loss is characterized by accentuation of the above symptoms and increasing instability of the circulation manifested by increasing pulse rate, orthostatic hypotension, diminished radial pulse volume and mottling of the extremities. Evidence of loss of tissue turgor may or may not be present. Further progression is accompanied by falling blood pressure to shock levels. The brachial artery pressure may fall to a point where it can no longer be recorded and yet the patient remains conscious. The general condition of the sensorium may fail

to reflect the degree of peripheral circulatory insufficiency. Indeed, a heightened facial coloring, because of a relative polycythemia that sometimes accompanies this type of sodium depletion does not reflect the usual facies seen in severe peripheral hypotension. Experimentally produced electrolyte shock has demonstrated that the cardiac output may drop to levels that ordinarily are incompatible with life. Apparently the attendant increase in general peripheral resistance is of such magnitude that vital organs are able to maintain a satisfactory blood supply for a short

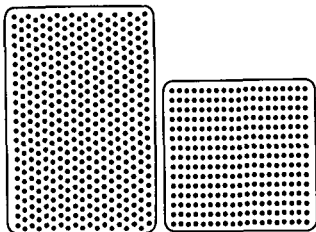


FIG. 360 Extracellular fluid volumetric depletion. *Left* Figurative expression of normal volume and sodium concentration of extracellular fluid as black dots. *Right* Volumetric depletion. The extracellular fluid volume is less than normal and there is a depletion of total body sodium. The sodium concentration in the remaining extracellular fluid will be normal when determined as milliequivalents per liter. Routine blood chemistries do not reflect volumetric loss of the iso-osmotic type and cannot be used to make the diagnosis.

period of time. This degree of vascular resistance has not been observed in traumatic or hemorrhagic states.

It is important to emphasize that serum sodium, chloride, potassium, and bicarbonate concentrations per ml may be entirely normal in volumetric disturbances. Reliance upon routine blood chemistries to reflect this type of sodium depletion is fallacious as well as hazardous. The blood non-protein nitrogen is usually elevated, however, and potassium levels may also be increased. The increased non-protein nitrogen has been related to diminished glomerular filtration rates observed in sodium salt depletion, but the significance of the occasionally observed elevated serum potassium is unknown. The ability of the kidney to clear potassium is not decreased. It may represent the transfer of intracellular potassium and electrolyte in response to such stress.

Hemoglobin concentration and hematocrit and serum proteins may all be elevated, the degree being directly proportional to the rapidity of the extracellular fluid loss. Urine output is diminished but the specific gravity is variable. Albumin, red

blood cells, white blood cells, and casts may be present in urine sediment.

Disturbances of Sodium Concentration of Extracellular Fluid. These are of two principal types: hyperconcentration or dilution of the sodium salts (osmolar disturbances) and aberrations in extracellular bicarbonate content (acid-base disturbances).

Osmolar Changes. Increased concentration of extracellular sodium (greater than 145 mEq.) may be observed when water is lost from the body faster than the kidney can excrete the necessary salt in order to maintain normal osmolar concentration. Withholding water when it is needed, high environmental temperatures, high body temperatures, and certain injuries and diseases of the central nervous system may be attended by such hyperconcentration of sodium salts.

More commonly, however, the opposite is observed during intestinal obstruction—that is, a lowering of the sodium level of extracellular fluid (below 136 mEq.). This is usually associated with a volumetric defect of extracellular fluid. Not only is there a diminution of total body sodium but there is a lowering of the unit concentration of sodium and usually also of chloride. If parenteral water is provided at a rate greater than the ability of the kidney to excrete the net excess or under circumstances when renal function is impaired, true water intoxication supervenes from dilution of the sodium salt concentration. In intestinal obstructions, however, the circumstances for true water intoxication rarely present themselves. A condition that clinically resembles true water intoxication may result from sodium depletion. This has been described by Moyer as "extracellular salt-water deficit with relative water excess." A source of parenteral water is necessary in order to lower the serum concentration of sodium during the creation of an acute sodium deficit from the loss of extracellular fluid. Leaf and Mamby have observed that up to a certain point water is excreted by the kidney in order to maintain normal relationship between extracellular salt and water. Due to adaptations which remain little understood, lowered extracellular fluid volumes apparently reach the point where osmolar integrity is sacrificed in order to maintain volume. As a result, renal excre-

tion of water fails to keep pace with parenteral administration and the serum level of sodium begins to fall. The resultant lowering of osmotic pressure of extracellular fluid is thought to permit the passage of water into body cells. The resultant symptomatology is due to cellular edema and usually first becomes clinically apparent because of edema of the central nervous system.

The initial symptom is usually mild disorientation followed by restlessness, muscle twitchings, delirium and even convulsions. Spinal fluid pressures are increased and after a short period of time can produce papilledema. Increased water content of the cells of the skin produces a hard non pitting fingerprint type of edema. Extra renal excretion of water may be attempted by salivation, lacrimation, diarrhea and vomiting.

The serum sodium is low as are the chloride and CO_2 content. Potassium levels are variable; the non protein nitrogen is elevated.

Hemoglobin, hematocrit and serum levels may reflect the plasma dilution. Urine flow is scanty and of variable specific gravity. Sediment may show evidences of renal tubular irritation observed in volumetric defects.

Acidosis and Alkalosis The bicarbonate concentrations of body fluids are of great importance and probably have a more significant role in electrolyte distribution and excretion than has previously been recognized. At normal serum pH of 7.4 dissociated carbonic acid ($\text{H}+\text{HCO}_3^-$) and base bicarbonate (90 per cent of which is $\text{Na}+\text{HCO}_3^-$) exist in a ratio of 1:20. The amount of dissociated carbonic acid is proportionate to the quantity of CO_2 dissolved in plasma (pCO_2) and therefore is under respiratory control. Any mechanism that increases the respiratory rate will lower dissociated carbonic acid by decreasing pCO_2 (respiratory alkalosis). Base bicarbonate concentration of extracellular fluid is under renal control and essentially represents the retention or excretion of sodium in defense of pH. The reciprocal relationship between base bicarbonate and dissociated carbonic acid (respiratory bicarbonate) is the most important extracellular buffering mechanism in the maintenance of normal pH and

is described in the Henderson-Hasselbalch equation

$$\text{pH} = 6.1 + \log \frac{\text{BHCO}_3}{\text{HHCO}_3}$$

The loss of sodium in excess of chloride (diabetic acidosis, pancreatic fistula) is accompanied by a decrease of HCO_3^- to BHCO_3 , because electrical neutrality must be maintained (metabolic acidosis) (Figure 3-8). Unless there is a compensatory loss of HCO_3^- as NH_4HCO_3 , the pH will fall. Respiratory compensation is accomplished by increasing the rate and amplitude of respiration, thus increasing the elimination of CO_2 and decreasing pCO_2 and dissociated carbonic acid ($\text{H}+\text{HCO}_3^-$). In conditions of marked sodium loss, the respiratory compensation is only limitedly effective.

The converse is true if chloride is lost in excess of sodium (pyloric obstruction). A relative increase of HCO_3^- to BHCO_3 takes place in order to maintain electric neutrality. Compensatory efforts to prevent an increase of pH will include slowing of the rate and diminishing amplitude of respiration so as to increase pCO_2 and dissociated carbonic acid ($\text{H}+\text{HCO}_3^-$). Although the renal defense of pH is initially supported by increased sodium excretion under such circumstances, this mechanism becomes diminishingly effective as a point is reached when sodium is conserved in order to maintain the volume of the extracellular fluid (Figure 3-9).

The great majority of acid base disturbances in intestinal obstruction are of the metabolic type that is they result from the loss of extracellular fluid, improper replacement solutions and starvation. Reduced to the simplest consideration, acid base disturbances of this type represent a loss or relative gain of sodium as compared to chloride. The correction of severe acidosis or alkalemia is the first priority in the treatment of any fluid imbalance state.

Potassium Deficits and Intestinal Obstruction Starvation loss of gastro-intestinal fluids, parenteral sodium salt administration, acidosis and alkalemia all contribute to a relative depletion of the body stores of potassium in obstructive states. Initial loss of potassium is of little impor-

tance but prolongation of the depletion factors can ultimately result in a serious loss.

Many of the symptoms of acute potassium deficit are similar to those of sodium. A significant loss of potassium may be masked by an extracellular fluid deficit and may become apparent only after the correction of the extracellular fluid deficit has been attempted.

There are four diagnostic considerations that should strongly suggest a potassium deficiency.

- 1 Severe weakness out of proportion to the general physical condition of the patient and to the seriousness of the clinical condition
- 2 Failure to obtain an adequate clinical response from sodium salt solutions
- 3 A refractory silent ileus
- 4 A refractory alkalo- or acidosis that has not responded to apparently adequate treatment

Serum potassium determinations are not entirely reliable since they may be within normal limits even though a deficit exists. Electrocardiograms are helpful in following the response to potassium treatment but they may otherwise give little information unless controls are available for comparison.

In summary, the clinical history is the best determinant as to whether a potassium deficit is present. As long as the urine flow is adequate, potassium salts should implement the parenteral fluids being used in intestinal obstruction.

PARENTERAL FLUID THERAPY

Parenteral fluids are used during intestinal obstruction for the following purposes:

- 1 Maintenance of an adequate urine flow and provision of water to cover insensible loss
- 2 Maintenance and repletion of external electrolyte loss
- 3 Repletion of the hidden loss of extracellular fluid (distributional shifts, extracellular fluid that remains in the body but has become physiologically ineffective)
- 4 Repletion of acute protein deficits and red blood cell loss
- 5 Implementation of nutrition
- 6 Correction and maintenance of the vital pH functions of body fluids

The degree of importance of these objectives depends upon the nature of the obstruction, the physical condition of the patient, and the sequence of events attendant on the illness. Because of these variables, the use of fluids will be discussed under the following headings:

- 1 Acute obstructions without significant extracellular fluid loss
- 2 Acute obstructions with significant extracellular fluid loss with and without osmolar and pH changes
- 3 Chronic intestinal obstruction

Acute Obstructions Without Significant Extracellular Fluid Depletion

These for the most part represent *maintenance* problems. Certain considerations peculiar to the postoperative period have also been included in the subsequent discussion.

Water. Previous reference has been made to the observation that during intestinal obstructions the loss of water and the loss of electrolyte do not necessarily occur at the same rate. It is necessary to differentiate between water administered as a sodium salt solution for repletion or maintenance of the extracellular fluid volume and water given to cover obligatory sensible and insensible loss and insure a good urine flow. The administration of an isotonic 0.9 per cent sodium chloride solution as a source of *water only* is a pernicious practice which affords little good and much potential harm. Under these circumstances the sodium chloride must be excreted in the urine in higher concentration than administered before water is available to cover obligatory losses. Operating at maximum concentration for sodium chloride, the urine contains only 2.0 per cent of this salt (320 mEq). This means that under the most favorable circumstances about 500 ml of water are made available for obligatory water needs from each 1000 ml of 0.9 per cent sodium chloride. The remaining water is used to excrete the excess sodium salt.

Water *per se* is best provided by the intravenous administration of a 5 to 10 per cent carbohydrate solution. The subcutaneous administration of this solution is less desirable because of osmotic diffusion of body electrolyte into the area of injection.

ous as water cannot be excreted by the damaged kidney. The resultant overexpansion of the circulating blood volume inevitably results in pulmonary and/or cerebral edema. Water intake must be sharply curtailed until healing of the nephron damage permits urine excretion to be resumed. The management of acute failure in the previously intact kidney has been the subject of many excellent papers including those by Blackburn and his co workers. Oliver and his co workers and Maluf, which should be consulted for details of treatment.

Maluf, which should be considered as a treatment so generally recognized that careful outputs is important

1 Too rapid administration of carbohydrate and water solutions. Such procedure speedily satisfies immediate water needs with the result that the excess water is excreted almost as fast as it is given. The circulation becomes a transport mechanism between the intravenous needle and the kidney with little utilization of the injected solution being accomplished excepting partial removal of the circulating carbohydrate.

2 Infusion of excessive amounts of carbohydrate and water The need of each patient is different depending upon environmental temperatures, hyperreflexia, extra renal loss, and ability to utilize intravenously administered water The routine use of 3000 cc of glucose and water per 24 hour period may be far in excess of the needs of certain patients particularly the aged

4 The mobilization of edema fluid
compensation burns peritonitis)
the recovery phase of the universal nephron

6 Failure of production of antidiuretic substances promoting distal tubular water absorption (diabetes insipidus)

Persistent diminished urinary output with associated low specific gravity (1010 or less) is very serious and should be regarded as evidence of acute renal damage until proved otherwise. Under these circumstances the forcing of parenteral fluids to increase urine flow is extremely dangerous.

adequate urine output means little unless it is carefully correlated with intake. A large urine output may actually be masking a negative water balance produced by the injudicious administration of intravenous carbohydrate and water solutions. In certain patients a vicious cycle may be set up due to the fallacy of covering the output of an intact kidney with intravenous fluids. In cases where urine volumes are compensated for by increasing amounts of intravenous fluids until 5000 to 10,000 cc of parenteral fluid are being given daily to cover the urinary loss. Cessation of excessive intravenous water administration will be followed by a sharp drop in urine output to a normal volume.

Electrolyte. Initial determination of serum sodium, potassium, chloride and CO_2 content as well as hemoglobin, venous hematocrit and serum proteins will provide a point of reference to aid in determining the significance of subsequent change in chemistries.

The minimal volume requirement of sodium salt replacement solutions can be empirically estimated by the sum of that removed by long tube suction and the estimated loss by vomiting. Determination of the amount of extracellular fluid lost or rendered physiologically ineffective during intestinal obstructions is not a simple measurement of the external loss. The hidden losses of sodium-containing fluids through pooling in the lumen of the obstructed gut and transudation into the intestinal wall and peritoneal cavity are undetermined variables that can be of vital importance. Adynamic ileus and mechanical obstructions of the terminal ileum are quite capable of producing a marked depletion of the effective extracellular fluid volume with little if any external loss by vomiting or suction. It is not uncommon to remove 2000 to 4000 ml of intestinal fluid from the distended gut during operations for small bowel obstruction. Because intestinal obstructions are invariably accompanied by a hidden loss of body fluids, *repeated evaluation by physical examination in order to detect the early signs of extracellular fluid depletion is essential.* Blood chemistries may be quite normal and therefore of little help under such circumstances.

What type of sodium salt solutions should be

given? Ideally, it would be desirable to obtain repeated daily laboratory analysis of the fluid being lost during obstructions as the day to day ionic constituency may change even with the tube in the same place. Such procedure is impractical and costly for routine use but may be necessary during the course of long and complicated obstructions. It has been our experience that the simplest solutions are the best.

In pyloric obstruction a volume for volume replacement of aspirated and vomited gastric fluids is carried out with U.S.P. Ringer's Solution. This solution not only has a greater quantitative number of chloride ions than sodium but actually carries a 40 per cent excess when compared to sodium and chloride in extracellular fluid (sodium 142 mEq—chloride 103 mEq). Only with great rarity has the administration of 0.9 per cent ammonium chloride been necessary to combat alkalosis and then only when the CO_2 content of serum has been greatly in excess of normal and tetany is present. Two grams of potassium chloride are added to each 1000 ml of administered Ringer's solution if the urine output is adequate (more than 800 ml per 24 hours with intact tubular concentrating ability). Pyloric obstructions unaccompanied by peritonitis are not characterized by clinically important hidden loss of fluid.

It is important to emphasize a singular complication of fluid replacement that is occasionally observed during pyloric obstruction. Hypersecretion and diminished absorptive ability accompany distention of the gut. Decompression usually diminishes hypersecretion and improves absorptive ability. In certain patients, however, it would appear that hypersecretion by the gastric mucosa continues during parenteral saline administration. The stomach becomes a second kidney and the major part of the sodium solutions being given to cover suction loss passes rapidly through the stomach wall and into the lumen only to be reaspirated, thus setting up a vicious circle. More and more sodium salt solutions are given to cover more and more removed gastric fluid until as much as 5000 to 10,000 ml per 24 hours of sodium salts are involved in this circle. Whenever the decom-

|| U.S.P. Ringer's Solution contains NaCl 8.6 gm, KCl 0.3 gm and CaCl 0.33 gm.

pressed stomach continues to put out more than 2000 ml per 24 hours by suction the status should be carefully reviewed to rule out a vicious circle of sodium replacement. The correction of excessive output/replacement of sodium salts is effected by temporary withholding of all saline solutions until the gastric secretion returns to normal. If further parenteral fluids are then indicated judicious saline replacement reinforced by whole blood transfusions is the treatment of choice.

Intestinal secretions lost from small bowel obstructions tend to parallel plasma in their ionic constituency. The chloride to sodium ratio may be almost equal in jejunal obstructions but decreases in the lower gut. It has been our experience that for the most part these losses can be matched volume for volume with U.S.P. Hartmann's Solution.** Certain patients may show increasing CO₂ content during this treatment. If such is the case then the electrolyte replacement can be accomplished with a solution made up of equal parts of Ringer's and Hartmann's solutions. If an adequate urine output is present these solutions should be further reinforced by the addition of 2 gm of potassium chloride for each 1000 ml of sodium salts given.

Hidden loss of fluid (distributional shift) can be most important in this type of obstruction. It is impossible to predict by any known laboratory technique the amount of such loss. It is therefore necessary to use clinical evaluation in order to follow the adequacy of sodium replacement. In dynamic ileus particularly the hidden loss of fluid may be of considerable magnitude. It is possible to develop a clinically severe extracellular fluid deficit because of intraluminal pooling without a single milliliter of gastro-intestinal fluid being lost by vomiting or intubation. If loops of small bowel are obviously distended on the abdominal scout film but no vomiting has ensued and if the long tube has not been passed and no clinically significant evidence of extracellular fluid depletion is present a minimum of 1000 ml of Hartmann's solution should be given and the patient closely watched for symptoms suggesting additional need.

** U.S.P. Hartmann's Solution contains NaCl 6.0 gm, Na₂HPO₄ 0.1 gm, KCl 0.3 gm and CaCl₂ 0.2 gm.

It is obvious that fluids removed by long tube suction do not represent the total loss of effective extracellular fluid volume. A minimum of 30 percent of measured vomitus and tube drainage should be added to the amount of sodium salts being given in addition to a volume for volume replacement of measured loss to cover hidden loss particularly if the obstruction is in the lower small bowel. Additional sodium salts may be necessary the need being gauged by clinical evaluation.

Blood Transfusions and Nutrition. Sodium salt depletion is accompanied by a progressive immobilization of plasma proteins which can in part be remobilized upon replacement of sodium salts. Because of attendant starvation and increased tissue catabolism during obstruction whole blood transfusions are of importance to aid in covering this acute protein deficit and to implement the circulating red blood cell mass. On an empirical basis 500 ml of blood should be transfused for each 3000 ml of sodium salts necessary for maintenance of the extracellular fluid volume. If peritonitis is attendant larger amounts of whole blood are necessary.

The problems of nutrition during intestinal obstruction are discussed in some detail in Chapter 11. Chronic Obstructions. In the well nourished patient the problems of maintenance of nutrition are not of primary concern during acute obstructions responding to treatment. Four to five days of starvation are well tolerated by the majority of previously undepleted individuals. The importance of the nutritional defect is directly proportional to the duration of the obstruction.

Strangulated and Vascular Obstructions. Because of severe abdominal pain and intractable vomiting strangulated obstructions tend to be seen early in the course of the disease. The hooklike picture seen early in strangulated obstructions is probably not due as much to loss of extracellular fluid as it is to imbalance of the autonomic nervous system from greatly augmented visceral afferent stimulation. Delay of operation is wisely taken in strangulated obstructions in order to stabilize the circulation provided the blood pressure has fallen below 100 mm of mercury. The hypotension of very early strangulated obstruction requires treatment which is firm support. Isolated strangulation

obstructions particularly of the terminal ileum can be associated with a severe extracellular fluid deficit. Hypotension under these circumstances is due principally to the loss of sodium salts and water. Correction of this deficit prior to operation is of the greatest importance as the electrolyte depleted subject poorly tolerates the trauma of operation.

Vascular obstructions due to thrombosis or embolism of the superior mesenteric vessels are not necessarily fatal catastrophes. Before operation is attempted the circulation must be made reasonably stable. Failure to restore circulatory stability will almost invariably result in an operative mortality.

Acute Obstructions with Significant Extracellular Fluid Loss

Volumetric Defect. This type of deficit is characterized by the loss of volume of extracellular fluid and a deficit of total body sodium is present. The unit concentration of sodium per milliliter of remaining extracellular fluid is within normal limits (Figure 360).

Case History

A 38-year-old white woman underwent total colectomy and anal ileostomy for congenital polyposis of the colon and malignant polypoid degeneration of the sigmoid colon. She received 1000 cc of whole blood during the operation and this was followed by 1500 cc of 5 per cent dextrose in distilled water. During the first 24 postoperative hours 500 cc of urine were excreted and 500 cc in intestinal contents were removed by long tube suction. The day after operation she was given 2000 cc of 5 per cent dextrose in distilled water and 500 cc of Hartmann's solution. During the same period 700 cc of urine were excreted 400 cc obtained by intestinal suction and 150 cc passed through the anal ileostomy. Forty-eight hours after operation the patient was observed to be lethargic but there was a definite suffusion of the face with heightened coloring (dehydration polycythemic flush). She was oriented and answered questions well. The hands were cold and the nail beds cyanotic. Filling of the veins on the dorsa of the hands was absent. The radial pulse was absent, and the brachial artery pressure could not be recorded. Tissue turgor was good.

The hemoglobin content was 16 gm. A specimen of urine revealed a specific gravity of 1.015, a 1+ reaction for albumin. The sodium content was 138 mEq, potassium 6.9 mEq, CO_2 54 vol per cent, chloride 85 mEq, and non protein nitrogen 77 mg per 100 cc. A diagnosis of severe extracellular fluid deficit, iso-osmolar in type as far as its chief clinical and laboratory considerations

with associated relative concentration of the red blood cell mass was made. During the next 12 hours she received 4000 cc of Hartmann's solution. At the end of four hours administration of this solution the brachial artery blood pressure had returned to 118/82 and filling of peripheral veins had returned. Upon completion of repair of the extracellular defect the hemoglobin content was 12.5 gm. Twenty-four hours later blood chemistry values were sodium 137 mEq, potassium 3.4 mEq, chloride 87 mEq, carbon dioxide 62 vol per cent, and non protein nitrogen 65 mg per 100 cc.

This patient presented the clinical and laboratory picture of a severe iso-osmolar volumetric defect of extracellular fluid that responded adequately to sodium salts.

The amount and type of sodium solutions to be given depends upon the clinical estimate of depletion, the CO_2 content and blood pH.

1 Normal CO_2 content and blood pH. Give 4 per cent of body weight of Hartmann's solution if hypotension is not present in the recumbent position. In the presence of severe peripheral oligemia and recumbent hypotension a minimum of 6 per cent of body weight of this fluid should be given. Consideration should be given to the relative fat content of the body. In the obese smaller amounts should be given and the clinical response noted. These are only guides and not rules for the correction of these deficits. Stabilization of blood pressure, return of color, warmth and venous filling to the extremities and establishment of a good urine flow permit reasonable assumption that the major part of the defect has been corrected. Because of oliguria the administration of larger amounts of potassium should be withheld until a good urine flow has been re-established.

2 CO_2 content less than 18 mm (40 vol per cent) with normal or lowered serum pH. Correction initiated as per (1) except that 300 ml of a one third molar sodium lactate solution is given simultaneously by vein in the opposite arm. A one sixth molar sodium lactate solution is isotonic with plasma. Because a one third solution is hypertonic it should be given slowly (250 ml/hr) and repeated rectal temperatures taken in order to prevent excessive hyperpyrexia sometimes observed with the administration of hypertonic sodium salts.

3 CO_2 content above 27 mm (63 vol per cent) with normal or increased serum pH. Correction

initiated as per (1) except that Klinger's solution is substituted for Hartmann's.

4. **Blood transfusions.** During the correction of volumetric defects blood transfusions may not be necessary. They aid however in insuring the repair of the defect particularly in elderly patients who may inefficiently mobilize their disappeared plasma proteins or in those patients who after reconstitution of the extracellular fluid volume reveal a low hemoglobin concentration.

In the presence of severe prolonged electrolyte shock it may be desirable to administer a 1.3 per cent solution of sodium bicarbonate rather than lactate as the lactate radical is inefficiently metabolized by a hypoxic liver and the sodium therefore is not available. The amount to be given is approximately 60 mEq. of NaHCO_3 per kilo of body weight for every millimole that the CO_2 content of serum is below 27 mm.

After correction of a volumetric loss attention should then be directed toward accurate maintenance of the extracellular fluid volume in order to avoid repetition of a potentially dangerous mistake.

Osmolar Defects. There are two principal types of lowered sodium serum concentration in surgical patients.

1. An internal adjustment in response to stress

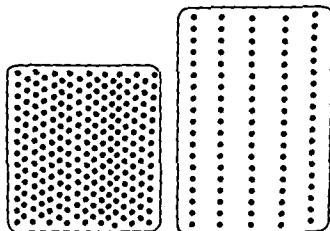


FIG. 361. Water intoxication. *Left*: Normal volume and sodium concentration of extracellular fluid. *Right*: Increased volume and sodium dilution produced by (a) intake of water at a rate greater than it can be excreted by the kidney (true water intoxication) or (b) administration of oral or parenteral water during periods of diminished renal function or acute damage to the kidney.

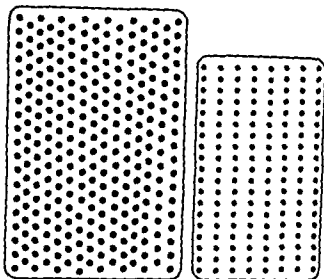


FIG. 362. Water intoxication secondary to acute sodium salt loss. *Left*: Normal volume and sodium concentration of extracellular fluid. *Right*: Loss of extracellular fluid volume and dilution of sodium from the administration of salt-free parenteral water. During periods of acute sodium salt loss the incorrect replacement of loss with carbohydrate and water solutions eventually results in the retention of water and extracellular hypo-osmolarity. Just why the osmotic integrity of extracellular fluid is sacrificed under these circumstances is not known. It may be related to some volumetric deficiency of the extracellular fluid.

This is commonly seen after operation, severe trauma, and during severe debilitating disease. For the most part these probably represent the normal physiology of disease or injury and are little understood.

2. Excessive or incorrect administration of carbohydrate and water in the presence of an extracellular volumetric defect or diminished renal ability to excrete water. The reason that the defense of extracellular fluid tonicity is sacrificed in order to defend volume is not understood but it has been related to some unknown volumetric function of the extracellular fluid. When accompanied by signs and symptoms of water intoxication this type of sodium deficiency is most serious and requires prompt treatment (Figure 361 and 362).

The following case history is an example of a severe hypo-osmolar and volumetric sodium deficiency.

Case History.

A 54-year-old Negro woman was admitted for review

gation of cancer of the lung. Three days after a lumbotomy he began to complain of left upper quadrant pain, refused to eat, and began vomiting. Examination of the abdomen showed some tenderness in both lower quadrants. A long tube was placed and the patient maintained on parenterally administered fluids: 2000 cc of 5 per cent dextrose in water, 1500 cc of Hartmann's solution and 1000 cc of Ringer's solution with 2 gm of KCl were administered. During this period 650 cc of urine were excreted with repeated specimens showing a high specific gravity. The value for serum amylase was 95 units, the nonprotein nitrogen, 69 mg per 100 cc, and the CO_2 , 48 vol per cent.

During the next 24 hours her condition became rapidly worse. Abdominal examination revealed progressive tenderness, rigidity and tenderness and moderate rigidity. Urine output was inaccurately determined but 600 cc of pink brown material had drained through the long tube. A diagnosis of an acute surgical abdomen, probably mesenteric vascular occlusion, was made. At peritonitis the right colon and all but the proximal 12 inches of the small bowel were seen to be either frankly gangrenous or pre-gangrenous. The infarcted bowel was resected and continuity of the intestinal tract re-established by anastomosis of the proximal part of the jejunum to the left side of the transverse colon. 1000 cc of whole blood were administered during the operation which was tolerated surprisingly well.

During the first 24 hours postoperative period he became progressively disoriented and muscle twitchings developed. Extreme restlessness necessitated restraint. 2000 cc of 5 per cent dextrose in water and 1000 cc of Hartmann's solution were administered. 400 cc of urine were excreted and 700 cc of drainage obtained through the Cantor tube. The following day by gavage received only 200 cc of dextrose in water were administered. The serum sodium was 130 mEq and potassium 3.6 mEq. She became incontinent of liquid rectal fecal drainage.

Progressive oliguria was approaching anuria by the sixteenth postoperative hour. The face was ashen. Peripheral pulses and venous filling were absent. Brachial artery pressure was absent. Examination of the blood revealed hemoglobin 14 gm, sodium 113 mEq, chloride 75 mEq, potassium 4.8 mEq, CO_2 63 vol per cent and nonprotein nitrogen 137 mg per 100 cc.

Dramatic results followed rapid administration of 1000 cc of a 2 per cent sodium chloride solution. The blood pressure returned to 118/86, the radial pulse was 80, urine output sharply increased and there was marked improvement in the restlessness and disorientation. After completion of administration of the hypertonic saline solution the blood chloride was 101 mEq and CO_2 43 vol per cent. Repair of the extracellular defect was then continued with 2000 cc of Hartmann's solution, 500 cc of whole blood and 1000 cc of 5 per cent dextrose in water. During the following 36 hours the patient excreted 1400 cc of urine and blood chemistry values at that time were sodium 144 mEq, chloride 102 mEq, potassium 3.9 mEq, CO_2 56

vol per cent, calcium 9.7 mg per 100 cc, and nonprotein nitrogen 168 mg per 100 cc.

In the order of their priority the following are the therapeutic targets in hypo osmolar deficiencies:

- 1 Correction of severe symptomatic acidosis or alkalosis
- 2 Restoration of normal osmolality to the remaining extracellular fluid
- 3 Correction of the volumetric defect. The three tend to overlap. They apply only to metabolic defects.

1 Normal CO_2 content and blood pH. Give 500 ml of a solution made up of equal parts of 2 per cent NaCl and one third molar sodium lactate. In the other arm 500 ml of whole blood is started. After completion continue the repair with Hartmann's solution to correct the volumetric defect unless little improvement has been noted in restlessness and disorientation. Further hypertonic salt may be indicated but the rate of administration should not exceed 250 ml/hr and repeated rectal temperatures should be taken to prevent excessive hypervolemia.

2 CO_2 content less than 18 mm (40 vol per cent). Give 500 ml of a one third molar sodium lactate with simultaneous administration of whole blood in the other arm. With improvement of signs of water intoxication attention can then be directed toward correction of the volumetric defect with Hartmann's solution.

3 CO_2 content greater than 27 mm (63 vol per cent). Give 500 ml of 2 per cent NaCl and simultaneously 500 ml of whole blood. Continue repair of Ringer's solution.

It should be recognized that recommended amounts of hypertonic solutions are only guides as administration may have to be slowed, discontinued or greater amounts administered depending upon the clinical response. As the signs of water intoxication abate and urine flow picks up correction of the volumetric defect can be initiated. Attention should then be directed toward accurate maintenance of water and salt needs.

Whole blood transfusions are essential to insure the repair of a hypo osmolar sodium defect. The amount to be given depends upon the hemoglobin

level venous hematocrit and measured blood volume

Chronic Intestinal Obstruction

Prolonged adynamic ileus, acute adhesive peritonitis, abdominal carcinomatosis, dehiscence of abdominal wounds or intestinal anastomoses, edema of anastomoses, mechanical obstruction with proximal small bowel fistulas and recurrent obstruction following recent operation for intestinal obstruction are among the more common causes of prolonged obstructive decommissioning of the gastro intestinal tract. Ideal treatment of course consists of restoration of the continuity of the fecal stream. Operation may be thought to be inadvisable or deferred because of the patient's poor physical condition, the improbability of being able to accomplish an effective release of the obstructive mechanism or previous operative failures to correct the obstruction. These patients present difficult problems not only in the maintenance of protoplasmic integrity but in preparation for any contemplated operation.

The maintenance of the water and salt needs of these patients initially may not present too difficult a problem. However as the number of days increase during which they are being maintained on parenteral fluids, the efficiency of such treatment begins to deteriorate. The problem then becomes one of essentially trying to maintain adequate nutrition.

The administration of parenteral water, salt, vitamins, glucose, amino acids and fat no matter how skillfully done cannot adequately be substituted for a normal diet. The maintenance of the nutritional need until the time the obstruction spontaneously resolves or the optimal time for operation becomes reasonably apparent is to walk a surgical tightrope between the devil of exposing the patient to unnecessary further depletion by dependence upon conservative treatment which ultimately fails and makes operation even more hazardous because of such delay and the deep blue sea of operating before maximal preparation has been achieved or under such circumstance that the operation proves to be a middle-some surgery. There are no dogmatic criteria which can be outlined. Only years of experience with these difficult

cases can keep the inevitable errors in judgment at a minimum.

Treatment

1 **Water and Salt** The same principles apply as in acute intestinal obstruction. Prolonged parenteral fluid therapy, particularly when sodium salts are being administered is conducive to potassium depletion. From 2 to 6 gm. of potassium salts should be administered daily.

2 **Blood and Plasma Transfusions** A contracted blood volume measurable by conventional means is often found in these patients. Normal or even increased blood volumes may be found however. These are usually accompanied by a decrease in the circulating red blood cell mass and total serum proteins. The liberal transfusion of blood except in the very elderly is of great benefit. Normal hemoglobin levels mean little. Even with no obvious loss of blood it has been observed that 1000 to 2000 ml. of blood can be transfused in these patients without significant increase in normal hemoglobin levels. Daily plasma transfusions of 200 ml. have been advocated to cover acute protein deficits after restoration of the red blood cell mass. The use of plasma as a source of protein for nutritional purposes may be expensive and relatively inefficient. As a source of amino acids it has not proved to be particularly satisfactory in our hands.

3 **Carbohydrate** A minimum of 200 gm. should be given daily to prevent ketosis and to aid in the sparing of both endogenous and exogenous protein metabolism. Carbohydrate in excess of 200 gm. per day apparently has no further effect upon endogenous protein metabolism.

4 **Amino Acids** These substances should have their maximum effectiveness in conditions producing chronic intestinal obstruction. The repletion of body proteins by intravenous amino acid is indeed difficult if not impossible but if minimization or neutralization of a negative nitrogen balance can be achieved they are of benefit. The necessity for slow administration and the large volumes of water concomitantly administered make the administration of more than 3000 ml. per 24 hours impractical. Furthermore it is necessary to avoid excessive deamination by the administration of large amounts of energy producing calories. A

minimum of 400 gm of carbohydrate daily would seem essential. For the long term administration of 10 to 20 per cent carbohydrate solution it is necessary to place a small polythene catheter in a large vein such as the superior or inferior vena cava so that large amounts of carbohydrate can be given without producing phlebitis and venous thrombosis is invariably a complication when small veins are used. Recent investigative work by Christensen and his co-workers have suggested that if patients are preloaded with carbohydrate solutions and then amino acids given without carbohydrate a better utilization of amino acids is obtained. Although not confirmed in sick surgical patient such observations are of interest. The amount of intravenous water necessary for this technique may prove to be impractical, however.

5. Intravenous Fat. Although this is theoretically the best method of giving energy calories little benefit from their combined use with carbohy-

drate and amino acid solutions has as yet been observed. The problem of stability of these solutions in long term storage has not been satisfactorily worked out.

6. Vitamins. It has been pointed out by Hayes that under conditions of stress the requirements for vitamins may be greater than previously anticipated. Ample amounts of commercial water-soluble vitamins should always be added to these parenteral fluids to implement their utilization.

7. The Problem of Hypotonicity. It should be remembered that the normal for disease states is not necessarily that found in health. As I. D. Moore has pointed out following operation or trauma the serum sodium may be low even though the total body sodium is unchanged (Figure 363). Furthermore, during long periods of oral depriva-

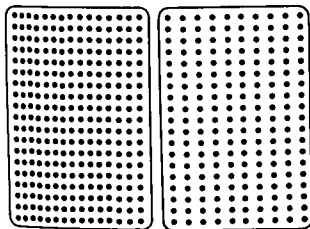


FIG 363 Physiologic hyponatremia following operation or severe injury. *Left*: Normal volume and concentration of sodium salts of extracellular fluid. *Right*: Following operation or injury the extracellular fluid may remain the same, increase or decrease in volume. A lowering of the sodium concentration is often observed. This is related to an internal shift and the serum concentration of sodium may be lowered even though the total body sodium remains the same or may actually be increased from the administration of sodium salts. Attempts to correct such physiologic hyponatremia with large volumes of water and sodium salts are relatively ineffective and may be the source of deleterious effects from overexpansion of the extracellular fluid. This condition is best left alone and allowed to undergo spontaneous correction with resumption of normal intake of food and activity.

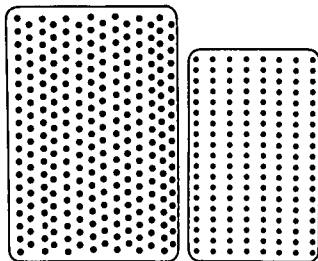


FIG 364 Chronic hypo-osmolality. *Left*: Normal volume and sodium concentration of extracellular fluid. *Right*: Contraction of volume and diminished concentration of sodium. This condition is different from that seen in Figure 362 in that it occurs over a period of days or weeks. The tenacity of body fluids falls as does the volume. The symptoms of water intoxication do not dominate the clinical picture despite a lowering of sodium to 120-125 mEq in serum. Attempts to restore normal concentration of serum electrolytes by the administration of large volumes of water, sodium and potassium salts will be ineffective. The main problem under these circumstances is malnutrition. The restoration of normal concentrations of serum electrolytes will be seen only after the proper oral intake of all elements of protoplasm is resumed.

tion there may be a gradual lowering of the tonicity of the extracellular fluid and probably also that of the intracellular fluid compartment. These represent stress adjustments and may not be abnormal for the condition that they represent. Attempts to correct such lowered tonicity of body fluids by large amounts of water and sodium and

potassium salts will not be effective and will only result in edema formation and further lowering of extracellular proteins. Under these circumstances the normal concentrations of serum electrolytes will be achieved only when an adequate oral intake is resumed, thus providing natural ingests of all of the essential elements of protoplasm (Figure 364).

DIVERSIONARY PROCEDURES

The diversionary procedures found useful in the management of the obstructed bowel are enterostomy, cecostomy and colostomy.

ENTEROSTOMY

Enterostomy has been used for a great many years to treat intestinal obstruction. This procedure was used extensively by surgeons not only in the treatment of all types of mechanical intestinal obstruction but also for paralytic ileus. As late as 1933 Ochsner and Gage, writing on the management of adynamic ileus, noted that a single enterostomy might fail to accomplish the necessary decompression because in this disease the propulsive power of the bowel was lost so that decompression would be limited to the specific loop of bowel drained by the enterostomy. For this reason they suggested that if the indications were present several enterostomies might be used in order to drain the small bowel more satisfactorily. They noted however that an enterostomy was not a cure all and that the most one could expect from it in cases of paralytic ileus was decompression of the segment of bowel which in turn might prevent interference with the blood supply to the bowel musculature and thus favor the return of intestinal movement. Ochsner and Gage emphasized the fact that enterostomy, even though of value in late cases of paralytic ileus, should not be used until all other remedies had been tried and found wanting. In 1931 Dixon of the Mayo Clinic suggested enterostomy at two or three points in the distended or obstructed portion of the intestine when the obstruction appeared in the distal jejunum or proximal ileum. This was performed by several incisions and two or three stab wounds

were required to bring the enterostomy out through the anterior abdominal wall. In some cases two enterostomies proximal to the point of obstruction were advocated for decompression purposes with a third enterostomy distal to the point of obstruction for feeding purposes. High jejunal enterostomy was advocated to treat high small bowel obstruction and was believed to be the solution to this problem of obstruction. Orr and Harden demonstrated however that the jejunal fistula produced by the enterostomy in this area produced the same changes in the chemical constituents of the blood as does a duodenal fistula. This is a dangerous situation because electrolyte alterations may prove rapidly fatal. For this reason unless the obstruction is found in the jejunum enterostomy is to be avoided. In an experimental study Hayden and Orr demonstrated that experimental animals treated by jejunal enterostomy lived a shorter period of time than those animals in which the obstruction went untreated. They also demonstrated that jejunal enterostomy in obstructed dogs resulted in death in two to five days and that all these dogs showed a decrease in the blood chlorides and a rapid rise in urea and non protein nitrogen. They consequently concluded that jejunal enterostomy in these experimental animals with high obstructions not only did not prevent the chemical changes in the blood typical of acute obstruction but that jejunal enterostomy tended to shorten life. The same observation was made by Moore on a clinical basis.

Methods of Enterostomy

Many methods of draining the proximal distended bowel by enterostomy have been devised

The method of Witzel was the method of choice for many years. This method consists of burying a small tube in the inverted wall of the small intestine. The loop of bowel selected for enterostomy is brought up, well packed off with laparotomy sponges, and then a drainage tube is laid along its antimesenteric edge. This tube is then covered with bowel wall by 000 interrupted sutures of silk or cotton. An opening is then made in the bowel at the end of the tube and the end of the tube inserted into the bowel. A purse string is then used to close the point of entrance of the drainage tube into the bowel. The drainage tube is then brought out through a stab wound in the side.

This technic was modified by Mayo. The modification consists of bringing the drainage tube through a piece of omentum before bringing it out through the stab wound in the abdominal wall. The omentum effectively seals the enterostomy opening in the bowel. With this technic the fistula usually closes spontaneously when the tube is removed if there is no obstruction distal to the enterostomy opening.

Gamble proposed a method of obstructive enterostomy to decompress the bowel in cases of intestinal obstruction. By means of obstructive enterostomy, Gamble prevented the contents of the obstructed bowel from entering the healthy bowel and also provided for the gradual evacuation of the contents of the obstructed bowel. To accomplish obstructive enterostomy, Gamble proposed the use of an enterostomy tube of sufficiently large caliber, preferably about half an inch in diameter. When a tube of this size is introduced into the proximal loop of bowel and held in place with superimposed purse string sutures, the lumen of the bowel leading away from the obstructed portion is completely closed and all the contents of the bowel are drained out through the large caliber enterostomy tube. This material is not drained off immediately. Generally a small amount flows when the tube is introduced. Following this, there is usually a period of 24 to 96 hours before much more drainage occurs. This is the latent period during which the peristaltic activity is being reestablished and atony of the bowel is being overcome. With the recovery of the physiologic function of the bowel, peristalsis begins and large

amounts of intestinal material are evacuated by the tube. In all cases in which obstructive enterostomy is performed, Gamble uses the open wound method of treatment because of the infection in the incision through which the tube is brought. He noted that regardless of the meticulousness of the technic, it is practically impossible to prevent infection of the operative wound because of the rich intestinal bacterial content associated with obstruction. By leaving the incision open and closing it secondarily, he has reported little or no trouble. Gamble reported that the obstructive enterostomy method and the liberal use of antibiotics with the restoration of electrolyte balance, oxygen, and the usual methods of treatment have resulted in a mortality rate of 7.9 per cent for 113 consecutive cases of intestinal obstruction. All types of acute small bowel obstruction were represented in this series. At the present time, this type of operative enterostomy is practically obsolete. In very unusual circumstances, however, the method may be used with profit as a temporary measure. The following case report is a good example of an occasion when this procedure proved effective.

Case report

H. M., a 40-year-old adult white male, was operated on for acute appendicitis. At the time of surgery, a ruptured gangrenous appendix was found with the area of appendiceal gangrene extending down to the cecum. There was marked intestinal distention at the time of surgery. Following removal of the appendix, the surgeon considered it desirable to decompress the bowel. For this purpose, he introduced a small catheter through the appendiceal stump and threaded it into the terminal ileum, bringing the catheter out through a stab wound in the side. Following surgery, the patient developed a local peritonitis and paralytic ileus. The ileus became progressively worse and the condition of the patient rapidly deteriorated. To treat this, the surgeon performed an ileal enterostomy using a loop of ileum 4 feet from the ileocecal valve. The type of enterostomy performed by the surgeon was externalization of the ileal loop and the insertion of a small catheter into its proximal end. Following this, the intestinal distention remained unrelieved. It was obvious that the patient had a fulminating peritonitis as a result of a leak at the site of the appendicostomy. When we saw this patient, his condition was critical. There was marked intestinal distention, rigidity, and marked tenderness in the lower right quadrant. A foul drainage exuded from the incision through which the appendicostomy had been made. There was no drainage from the ileal enterostomy. It was a surprise that the patient was devoid of peritonitis despite leakage from the appendicostomy. Cautious obstructive enterostomy

tomy procedure was utilized under these circumstances. A large caliber rubber tube was introduced through the ileal enterostomy and the tube was fastened well along the proximal limb. By this maneuver all the intestinal contents drained through the large caliber tube, the distal limb of the enterostomy being completely obstructed. On the second day after institution of 6 ambles of tractive enterostomy, 3000 cc of liquid intestinal content were drained through the tube. The patient improved markedly. The leakage through the appendicostomy was reduced to a minimum and the patient eventually overcame the acute infectious process. Further elective surgery was deferred to a later date.

There is little doubt that in a case of this type obstructive enterostomy was a life-saving measure in that it permitted the free drainage of intestinal contents proximal to the point at which leakage had occurred and at the same time diverted the fecal stream effectively so that little or none came through the leaking appendical area. Situations of this type are rare. In all our experience in the management of intestinal obstruction this was the first instance in which this problem arose. Complications of this type can be expected to occur less often as the surgical knowledge of operating surgeons throughout the country increases. The avoidance of appendicectomy as well as ileal enterostomy would have made the procedure of obstructive enterostomy unnecessary. We do not believe that procedures of this type including obstructive enterostomy should ever be used except in extremely rare instances.

Disadvantages of Enterostomy

Enterostomy is not as trivial a procedure as it was thought to be in the past although there is no question of its effectiveness in certain limited instances. The following disadvantages have been noted:

1. There is always a possibility of leakage with a resultant peritonitis regardless of the expertness with which the procedure is performed.
2. A persistent fecal fistula may cause a fatality when the closure of the enterostomy is attempted.
3. At times the fistulous tract closes only to reopen spontaneously some months later with wound disruption.

4. If the small bowel has already become atonic the operative procedure of enterostomy is useless because only that one loop of bowel will become emptied.
5. Whenever enterostomy is performed secondary operation should be done as soon as possible because the small bowel may perforate at the point of obstruction even though the bowel proximal to it had been decompressed by enterostomy.
6. One of the greatest blunders that can be made is to perform an enterostomy in the presence of a strangulating obstruction. When the enterostomy is made above the point of strangulation the bowel proceeds to perforation and peritonitis below.
7. Once the obstruction is released in a mechanical intestinal obstruction there is no point in performing an enterostomy because the bowel can empty itself normally.
8. Local or general peritonitis may be caused by enterostomy.
9. Wound infection is common following enterostomy.
10. The enterostomy may fail to relieve the obstruction. Digestion of the abdominal wall may occur with the use of jejunal enterostomy. This is the result of leakage.
11. When high jejunal enterostomy is used there is often leakage about the tube and the formation of a high intestinal fistula. This results in a loss of the water, sodium and potassium which are essential for the survival of the patient.

All these reasons plus the newer methods of obtaining the same result without enterostomy have relegated this procedure to the limbo of the past. Thus at present we believe that enterostomy should rarely be performed. Its use should be limited to those patients in such poor condition that nothing else can be done.

CECOSTOMY

One of the most important factors in colonic surgery has been the widespread acceptance of the concept that decompression of the colon by surgical means is an important aid in restoring the devitalized patient with a greatly distended

bowel as a result of malignant disease to a state approaching physiologic balance. When such decompression was required cecostomy was most commonly used in earlier days.

There are many surgeons who believe that with any degree of intestinal distention due to obstruction of the colon it is better to do a blind cecostomy than to explore the abdomen during the first operative procedure. Our experience is well as that of many surgeons in this country and abroad has been that with the relaxation obtainable by the use of spinal anesthesia or intratracheal anesthesia and curare there are very few cases in which it is too dangerous to carry out a gentle manual exploration by means of a transverse incision. In this way the presence of multiple primary carcinomas in the colon can be found. In addition one can determine whether or not there is liver metastasis whether the new growth is fixed or mobile and whether it has spread to the peritoneum. In those cases in which distention of the colon is so marked that the patient is threatened with possible perforation of the cecum one should not hesitate to insert a needle or a Poole suction tip into the most distended portion of the cecum. This is a safe and helpful procedure if the cecum has been packed off carefully to avoid soiling and an encircling purse string suture has been placed in the cecum at the point of puncture. The purse string can be snugged about the suction tip to avoid leakage. When the suction tip is removed the purse string is tied thus closing the opening made.

Indications for Cecostomy

1 The chief indication for cecostomy is as a decompressive measure for those growths in the upper ascending colon and at the hepatic flexure causing obstruction.

2 Cecostomy is commonly used as a supplementary procedure with obstructive resection of the left colon.

3 Cecostomy has been used successfully as a supplementary procedure with resection of the colon and anastomosis.

4 Cecostomy is used by many surgeons as a decompressive measure for intestinal obstruction secondary to carcinoma of the left colon or rectum.

5 Cecostomy may also be used as the first stage

of a planned two-stage operation for resection of the colon.

Technics for Performing Cecostomy

Various techniques for the performance of cecostomy have been proposed. In general these fall into two large groups—the tube cecostomy performed within the abdomen and the exteriorization type of cecostomy.

Tube Cecostomy. Surgeons generally agree that tube cecostomy is an inadequate decompressing procedure and that an exteriorized cecostomy is a much better procedure whenever it can be performed. When the cecum is fixed so that exteriorization cannot be used a tube cecostomy is indicated. The cecum should be well packed off a purse string suture of 000 chromic placed at the anterior tenial band, an opening made within the purse string loop and a suction tip inserted into the cecum. By so doing the cecum can be safely decompressed. A large size catheter or rubber tube is then inserted into the cecum through the opening made for the suction tip. The purse string suture is tied and a second purse-string suture of 000 silk is placed about the cecostomy tube. The edge of the rubber tube may be caught in the first purse string to fix it and prevent accidental removal. The cecum should then be tacked to the peritoneum of the anterior abdominal wall and the wound sutured about the cecostomy tube. If possible a piece of omentum should be wrapped about the cecostomy tube or the tube may be brought out through a hole in the omentum.

Exteriorization Cecostomy. The exteriorization type of cecostomy is being favored more and more by surgeons throughout the country as the most desirable type of cecostomy procedure. The technique of this procedure is relatively simple in those cases in which the cecum and right colon are not fixed. Some difficulty may be experienced in this performance in those cases in which the cecum and right colon are fixed in position. The exteriorization technique consists in simply exteriorizing as much of the cecum as possible and tacking its wall to the peritoneum. The cecum if distended may be punctured by a large caliber needle or a Poole suction tip before such exteriorization is made. A saline gauze sponge is placed about the cecum

and a dressing applied. The exteriorized loop should be examined twice a day for signs of distention. If this occurs the distended cecum can be decompressed by a large caliber needle. In the instances in which immediate decompression is indicated a large caliber rubber tube may be placed within the exteriorized loop of cecum immediately. In most cases a delay of 24 to 48 hours is preferable. In 48 hours the cecal wall has become so adherent to the abdominal wall that leakage into the peritoneal cavity is not likely.

The technic of exteriorization cecostomy has been modified by some surgeons in order to obtain better decompression, better cleaning of the colon proximal to the point of obstruction, and a reduction of the dangers associated with tube cecostomy.

Hunt's Modification of Exteriorization Cecostomy. Because of the difficulties and dangers associated at times with cecostomy, Hunt has abandoned this procedure in acute obstruction of the colon. He proposes a cecostomy procedure of his own. With his procedure he uses a special clamp with an opening in the distal end through which an 18 or 20 Fr catheter may be passed. The technic is as follows. If the colon is acutely distended the cecum is deflated by needle puncture after being well packed off. The deflated bowel is then grasped at the point of needle puncture with a flat no. tooth thumb forceps and traction is applied to it. The special colostomy clamp is then applied to the suspended cone of bowel. The abdomen is then closed around this protruding segment of colon. The tent of bowel within the clamp is then opened and an 18 or 20 Fr catheter is inserted into it. With this procedure only a small segment of colon is exteriorized but the segment is adequate for the relief of gaseous distention. If the colon contains chiefly liquid material and very little gas it may be deflated by the use of a suction tip so that the special clamp can be applied. The bowel is sutured around the catheter and is irrigated to deflate the colon. The clamp is removed in 48 to 72 hours or it may be permitted to remain in place until it sloughs off.

This method of performing cecostomy is proposed for use in those cases of acute obstruction of the colon as a result of malignant disease where

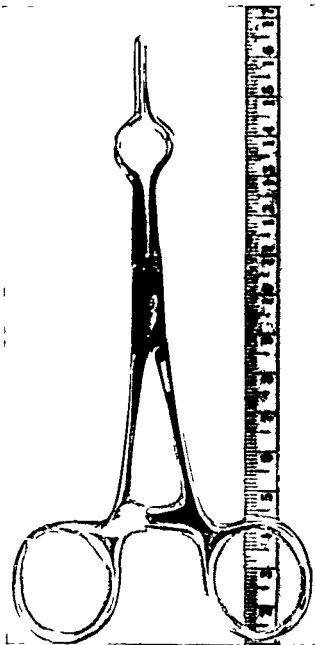


FIG. 365. Clamp used by Hunt to perform cecostomy.

subsequent resection is anticipated. It is not used where a complete diversion of the fecal stream is desired. Using this method an adequate amount of cecum is exteriorized which gives sufficient vent for gas to pass through the catheter.

Extraperitoneal Cecostomy. In this procedure a McBurney incision is made. The external oblique muscle is opened along the line of the skin incision. The internal oblique and transversalis mus-

howel as a result of malignant disease to a state approaching physiologic balance. When such decompression was required cecostomy was most commonly used in earlier days.

There are many surgeons who believe that with any degree of intestinal distention due to obstruction of the colon it is better to do a blind cecostomy than to explore the abdomen during the first operative procedure. Our experience as well as that of many surgeons in this country and abroad has been that with the relaxation obtainable by the use of spinal anesthesia or intratracheal anesthesia and current there are very few cases in which it is too dangerous to carry out a gentle manual exploration by means of a transverse incision. In this way the presence of multiple primary carcinomas in the colon can be found. In addition one can determine whether or not there is liver metastasis, whether the new growth is fixed or mobile and whether it has spread to the peritoneum. In those cases in which distention of the colon is so marked that the patient is threatened with possible perforation of the cecum one should not hesitate to insert a needle or a Poole suction tip into the most distended portion of the cecum. This is a safe and helpful procedure if the cecum has been pricked off carefully to avoid soiling, and an encircling purse string suture has been placed in the cecum at the point of puncture. The purse string can be suggested about the suction tip to avoid leakage. When the suction tip is removed the purse string is tied thus closing the opening made.

Indications for Cecostomy

1 The chief indication for cecostomy is as a decompressive measure for those growths in the upper ascending colon and at the hepatic flexure causing obstruction.

2 Cecostomy is commonly used as a supplementary procedure with obstructive resection of the left colon.

3 Cecostomy has been used successfully as a supplementary procedure with resection of the colon and rectum.

4 Cecostomy is used by many surgeons as a decompressive measure for intestinal obstruction secondary to carcinoma of the left colon or rectum.

5 Cecostomy may also be used as the first stage

of a planned two stage operation for resection of the colon.

Technics for Performing Cecostomy

Various technics for the performance of cecostomy have been proposed. In general these fall into two large groups—the tube cecostomy performed within the abdomen and the exteriorization type of cecostomy.

Tube Cecostomy Surgeons generally agree that tube cecostomy is an inadequate decompressing procedure and that an exteriorized cecostomy is a much better procedure whenever it can be performed. When the cecum is fixed so that exteriorization cannot be used a tube cecostomy is indicated. The cecum should be well packed off a purse string suture of 000 chromic placed at the anterior tenial band, an opening made within the purse string loop and a suction tip inserted into the cecum. By so doing the cecum can be safely decompressed. A large size catheter or rubber tube is then inserted into the cecum through the opening made for the suction tip. The purse string suture is tied and a second purse string suture of 000 silk is placed about the cecostomy tube. The edge of the rubber tube may be caught in the first purse string to fix it and prevent accidental removal. The cecum should then be tucked to the peritoneum of the anterior abdominal wall and the wound sutured about the cecostomy tube. If possible a piece of omentum should be wrapped about the cecostomy tube or the tube may be brought out through a hole in the omentum.

Exteriorization Cecostomy The exteriorization type of cecostomy is being favored more and more by surgeons throughout the country as the most desirable type of cecostomy procedure. The technic of this procedure is relatively simple in those cases in which the cecum and right colon are not fixed. Some difficulty may be experienced in the performance in those cases in which the cecum and right colon are fixed in position. The exteriorization technic consists in simply exteriorizing as much of the cecum as possible and tucking its wall to the peritoneum. The cecum if distended may be aspirated by a large caliber needle or by a suction tip before such exteriorization is made. A saline gauze sponge is placed about the cecum

and a dressing applied. The exteriorized loop should be examined twice a day for signs of distention. If this occurs the distended cecum can be decompressed by a large caliber needle. In those instances in which immediate decompression is indicated a large caliber rubber tube may be placed within the exteriorized loop of cecum immediately. In most cases a delay of 24 to 48 hours is preferable. In 48 hours the cecal wall has become so adherent to the abdominal wall that leakage into the peritoneal cavity is not likely.

The technique of exteriorization cecostomy has been modified by some surgeons in order to obtain better decompression, better cleansing of the colon proximal to the point of obstruction, and a reduction of the dangers associated with tube cecostomy.

Hunt's Modification of Exteriorization Cecostomy. Because of the difficulties and dangers associated at times with cecostomy, Hunt has abandoned this procedure in acute obstruction of the colon. He proposes a cecostomy procedure of his own. With his procedure he uses a special clamp with an opening in the distal end through which an 18 or 20 Fr catheter may be passed. The technique is as follows. If the colon is acutely distended the cecum is deflated by needle puncture after being well packed off. The deflated bowel is then grasped at the point of needle puncture with a flat no tooth thumb forceps and traction is applied to it. The special colostomy clamp is then applied to the suspended cone of bowel. The abdomen is then closed around this protruding segment of colon. The tent of bowel within the clamp is then opened and an 18 or 20 Fr catheter is inserted into it. With this procedure only a small segment of colon is exteriorized but the segment is adequate for the relief of gaseous distention. If the colon contains chiefly liquid material and very little gas it may be deflated by the use of a suction tip so that the special clamp can be applied. The bowel is sutured around the catheter and is irrigated to deflate the colon. The clamp is removed in 48 to 72 hours or it may be permitted to remain in place until it sloughs off.

This method of performing cecostomy is proposed for use in those cases of acute obstruction of the colon as a result of malignant disease where

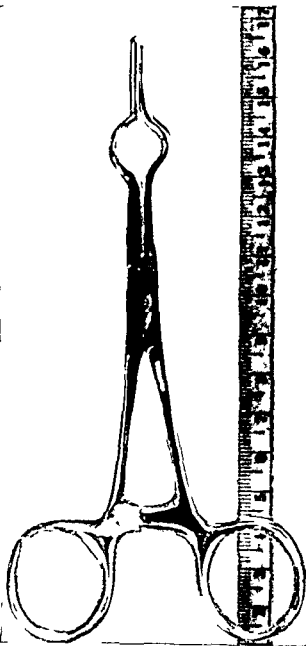


FIG. 365 Clamp used by Hunt to perform cecostomy

subsequent resection is anticipated. It is not used where a complete diversion of the fecal stream is desired. Using this method an adequate amount of cecum is exteriorized which gives sufficient vent for gas to pass through the catheter.

Extraperitoneal Cecostomy. In this procedure a McBurney incision is made. The external oblique muscle is opened along the line of the skin incision. The internal oblique and transversalis mus-

cles are then opened transversely along the line of their muscle fibers. The peritoneum is then pushed aside and the cecum is opened retroperitoneally. The opening in the cecum is made within a purse string loop placed in the cecal wall. A catheter of the desired size is then placed into the cecum and the purse string tied. A second purse string of 000 silk or cotton may be placed in the cecum about the first. Cattell suggested that a small bite be taken in the cecal wall by the suture to the catheter.

The advantage of this method is that the peritoneal cavity is not entered and a clean cavity is available for future surgery. Furthermore there is little or no danger of peritonitis. In the majority of cases this type of cecostomy closes spontaneously when the tube is removed.

A serious disadvantage is the danger of extensive retroperitoneal space infection. The fatty tissue in this space offers little resistance to infection so that infection in this space is all too common after retroperitoneal cecostomy. Such infections are apt to be very troublesome. In addition abdominal wall infection may create a serious problem. For these reasons this type of cecostomy is seldom used by American surgeons.

Adjunctive Procedures Supplementing Cecostomy Because cecostomy accomplishes decompression of the colon without completely defunctionizing it usually does not permit cleansing of the bowel proximal to the point of obstruction. Consequently in those cases where a decompression by cecostomy has been performed Allen and Welch propose the use of a cleansing procedure. A method using a Miller Abbott tube to accomplish this was suggested by Millet and Maybury. This method consists essentially of performing a cecostomy using a cecostomy tube 20 mm in diameter. Following this procedure several days of rest are permitted so that the colon may regain its tone and resume peristalsis. Four days later a small hole is cut in the superior aspect of the rubber drainage tube close to its attachment to the cecostomy tube. A Miller Abbott tube preferably of 15 Fr diameter is passed into the cecum and the balloon is inflated with 60 to 120 cc of air. A sufficient amount of slack is passed into the cecum to enable the tube to pass readily to the point of

obstruction. If the tube is so adjusted and the slack placed into the cecum is often as every 10 minutes the authors have seen the tip of the tube reach the sigmoid flexure of the colon within three hours. Irrigations of 1500 cc of normal saline solution or tap water twice a day through the Miller Abbott tube will cause back flushing of the colon from the point of obstruction and the drainage will flow from the cecostomy tube. Following each irrigation the Miller Abbott tube is placed on suction for 30 minutes to empty the bowel lumen of residual irrigating fluid and to maintain the bowel in a state of compression. Proponents of this method claim that it has certain advantages over cecostomy alone since actual cleansing as well as decompression of the colon may be obtained.

Brocq and Etidel described a method of decompressing the colon by intubating it with a single lumen tube weighted with mercury. They believe that their method is particularly useful in obstructions of the descending colon. The mercury tipped tube is introduced by means of a small cecostomy and repeated irrigations are carried out just above the point of obstruction. By combining intubation with irrigation at its lower end the large intestine may be completely emptied in aspiration which is carried out in the days following intestinal resection in order to prevent tension on the suture line.

Beardsley proposed a rather ingenious method for decompression of the cecum. To prevent the objectionable features inherent in cecostomy with spillage Beardsley has devised a suction trochar which is passed into the lumen of a 30 Fr rectal tube which is then introduced into the exteriorized cecum for the purpose of cecostomy. The dilated cecum after decompression is readily brought to the surface and sutured to the skin. The trochar is then removed after the catheter has been fastened in place within the cecum by means of a purse string. The trochar of the suction variety and readily permits decompression of the cecum so that the purse string may be placed easily and the cecum brought up to the abdominal wall.

Advantages of Cecostomy

- 1 The cecum is the most proximal portion of the colon and is most readily accessible so

that decompression may be obtained in this first portion of colon

- 2 Cecostomy is a relatively simple procedure which is carried out in an anatomic area familiar to the average surgeon
- 3 The operation can be performed under local anesthesia and requires only a small incision
- 4 The cecum is easily accessible and can be walled off from the rest of the peritoneal cavity
- 5 The cecostomy will not interfere with definitive resection of the left colon
- 6 Cecostomy is a direct approach to the part of the colon most vulnerable to perforation. If perforation has occurred it can be handled by suction and drainage as well as exteriorization
- 7 Cecostomy provides immediate decompression of the distended cecum and colon
- 8 Cecostomy may be used to prepare the patient's colon for definitive surgery
- 9 Cecostomy openings usually close spontaneously so that surgical closure if required is a relatively simple procedure

Disadvantages of Cecostomy

Although cecostomy will effectively decompress the colon it does not completely divert the fecal stream. For this reason it cannot really defunctionize the colon. Semisolid fecal material may accumulate in the colon distal to the cecostomy opening. Such intestinal content does not lend itself to evacuation by the cecostomy. Because the cecostomy is so far away from the point of obstruction in the sigmoid colon or rectum it may be difficult or at times impossible to thoroughly cleanse the colon between the cecostomy and the point of obstruction. Following cecostomy many surgeons have noted that at the time of surgery the ascending and transverse colon were so full of feces that definitive surgery was a difficult problem.

APPENDICOSTOMY

Appendicostomy is an operation which should be used rarely if ever. The appendix may be absent as a result of previous surgery; it may be atrophic; or it may be retrocecal in position. Even

when it is available, it presents a very small lumen which is inadequate for satisfactory drainage.

DIVERSIONARY ILEOCOLOSTOMY

This procedure is most useful in the management of those obstructions of the colon involving the cecum or ileocecal valve. Under such conditions cecostomy cannot be used properly. Although ileostomy has been used in the past in the management of this type of case it has generally fallen into disrepute. Most surgeons prefer ileotransverse colostomy as a diversionary procedure. However, because it is possible that with a competent ileocecal valve a closed loop obstruction may be permitted to remain behind it is preferable in such cases to transect the terminal ileum and perform an end to side ileocolostomy. The distal end of the transected ileum is then brought out through the abdominal wall as an ileostomy opening. A catheter can be threaded through this ileostomy opening into the ascending colon effectively decompressing it. The only indication for this type of diversionary procedure is in those patients in whom no surgical procedure for the removal of the neoplasm is contemplated or where it would be difficult to perform a transverse colostomy. Generally the removal of the primary tumor is worthwhile even in the presence of liver metastasis and especially if the continuity of the bowel can be restored. In the presence of acute obstruction the inflammatory reaction about the carcinoma may greatly increase the fixation and lack of mobility of the bowel. With proximal decompression this inflammatory reaction subsides to such a degree that what initially looked like a formidable fixed mass requiring extensive surgery actually requires a relatively simple operative procedure. For this reason this type of diversionary procedure has a limited range of usefulness.

DEFUNCTIONIZING TRANSVERSE COLOSTOMY

There has been considerable controversy as to the relative merits of cecostomy and colostomy as decompressive measures in acute large bowel obstruction. Despite the recent advances and the important role played by antibiotics it is universally recognized that surgical decompression must pre-

cede definitive surgery for all obstructive lesions of the colon associated with colonic distention. Under these conditions if the obstruction is in the left colon the surgeon must choose between two decompressive procedures: cecostomy or colostomy. With obstructions of the right colon he has no alternative but cecostomy.

In mechanical obstruction of the left colon particularly of the malignant variety there are two objectives. First the colon must be decompressed for the immediate relief of the obstructing process and second definitive surgical treatment must be directed toward the removal of the obstructing process. In those cases in which the obstruction is complete decompression of the colon at the earliest possible moment is of paramount importance.

It is universally agreed that the long intestinal decompression tube has very little place in obstructive lesions of the left colon. It is impossible to decompress obstructive lesions of the large bowel with long tubes except in those cases of obstruction close to or involving the ileocecal valve. In such cases since the distention will involve the small bowel proximal to the point of obstruction long tube decompression may be used. In all other types of obstruction of the colon with or without a competent ileocecal valve although the long intestinal tube may be used to decompress the small bowel if distended definitive and specific decompression of the colon can only be accomplished by cecostomy or colostomy.

I accept in those instances where the surgeon was not trained in the technique in most series of cases the morbidity and mortality rates were appreciably higher for cecostomy than for colostomy. It is quite evident that there is normally a certain amount of fecal contamination during the performance of the cecostomy whereas a colostomy can be performed with comparatively little spillage either during or after exteriorization. In the many series of cases reported comparing these two surgical procedures peritonitis proved to be the major cause of death following the surgical decompression.

For obstructive lesions of the descending colon and particularly the sigmoid colon recto sigmoid and rectum decompression may at times be ob-

tained by passing a large sized catheter past the point of obstruction through a sigmoidoscope. This must be done very carefully in order to avoid perforation. When this attempt at threading a catheter through the point of obstruction is successful it permits decompression and preparation of the patient for definitive surgery without preliminary surgical decompression (cecostomy or colostomy). However in those obstructive lesions that lie above the range of the sigmoidoscope or the extension in which the obstruction is impassable to a catheter of large luminal diameter surgical decompression is indicated. In addition in those cases in which accumulations of solid feces have occurred above the point of obstruction surgical decompression must be resorted to because the fecal material cannot pass through any rectal tube.

Types of Colostomy

There are many types of colostomy. The individual preference of the operating surgeon may determine to some extent the specific type of colostomy. The following types of colostomy are commonly used: the loop colostomy, the de Pezzer colostomy, the spur colostomy, the end colostomy, the ileotransverse colostomy, the colocolostomy and finally the pelvic colostomy.

Loop Colostomy. This type of colostomy is a safe, simple and easily performed method of completely defunctionizing the left colon. It may be performed under general or local anesthesia. It simply consists of making a transverse incision between the navel and the xiphoid, separating the rectus muscles, exteriorizing a loop of transverse colon and passing a glass rod through the transverse mesocolon thus holding the transverse colon suspended outside of the peritoneal cavity. A vaseline gauze is placed on and around the exteriorized loop and the incision in the abdominal wall is closed by interrupted suture. The exteriorized loop is irrigated twice a day. Distention of the loop is immediately corrected by a piruetting, a 20 gauge needle and syringe. Depending upon the urgency of the case the loop of colon is transected on the second or third day. At this time the loop of colon is well sealed to the abdominal wall so that danger of leakage or contamination of the wound is negligible.

de Pezzar Colostomy This type of colostomy consists of simply placing a de Pezzar catheter within the colon intra abdominally. The catheter is brought out of the abdominal wall through an opening in the omentum which effectively seals the tract when the catheter is removed. The de Pezzar catheter is sutured within the colon by using two purse string sutures—an inner one of 000 chromic and an outer of 000 silk or cotton. We do not consider this type of colostomy very desirable because of the semi-solid nature of the colonic contents. In addition although decompression is obtainable this type of colostomy cannot defunctionize or completely divert the fecal stream.

Spur Colostomy In this type of colostomy the proximal and distal loops of colon are sutured together for a variable distance. The loop is completely transected either at the time of exteriorization or several days later. This type of colostomy completely diverts the fecal stream and thus completely defunctionizes the left colon. It carries with it more possibilities for small bowel obstruction and is more complicated than the loop colostomy. In addition when a crushing clamp is used to cut through the septum between the loops in closing the colostomy there is a definite danger of peritonitis if the crushing clamp is applied too low or if a loop of small bowel has insinuated itself between the limbs of the colostomy.

End Colostomy This type of colostomy is used after abdominoperineal resection. It is a permanent type of colostomy and constitutes an artificial anus. The proximal end of the colon remaining after transection of the sigmoid colon is brought out through a stab wound on the side. The entire colon, rectum and anus distal to this are removed with the surgical specimen. A sufficient amount of colon is brought out through a stab wound so that there is no undue tension upon the vessels supplying the exteriorized segment of bowel. The stab wound should be made away from the anterior superior spine. This permits the comfortable use of a colostomy belt. When the swelling has completely subsided the exteriorized spur is trimmed down so that it lies flush with the skin much like an artificial anus—which it really is. This type of colostomy can easily be cared for

without the use of a colostomy bag. A vaseline gauze is applied to the opening, a Kotex pad is placed over it. The Kotex is held in place by a wide elastic belt. It takes a simple clean and easily handled dressing.

Ileotransverse Colostomy This type of colostomy is used in the management of obstructions of the right colon. It is to be followed by a second stage resection of the right colon. The procedure may simply consist of anastomosing the terminal ileum to the transverse colon thus circuiting the obstruction in the right colon. If the ileocecal valve is competent it may be to exteriorize the distal portion of ileum proximal end of transected ileum is anastomosed to-side to the transverse colon.

Colocolostomy This is a diversionary procedure which may be found useful as a temporary measure prior to a second stage operation. The procedure consists of anastomosing a portion of the transverse colon to the rectosigmoid as a diversionary procedure for obstruction of the left colon. This procedure does not divert the fecal stream completely and some intestinal content does enter into the bypassed loop.

Pelvic Colostomy This type of colostomy is reserved for those neoplasms in the pelvis which for various reasons cannot be resected. It is bad surgery to perform an emergency colostomy for a resectable obstructing lesion of the rectosigmoid because it embarrasses the next stage of the procedure and necessitates operating in a contaminated field with the operative site a mass of adhesions.

Advantages of Transverse Colostomy

There are many surgeons including those who believe that a transverse defunctioning colostomy is a safer and more efficient than a cecostomy except in the management of lesions of the right colon. From a surgical literature review decompression appears to have satisfactory results in approximately 30 per cent of cases with cecostomy while this was true in 3 per cent of the cases in which transverse colostomy was used. For obstructive lesions of the middle of the transverse colon we be-

a loop colostomy in the right transverse colon gives more complete relief from obstruction than any type of cecostomy. In addition it permits one to clean the bowel above the obstructing growth more efficiently.

The advantages of transverse colostomy over cecostomy may be tabulated as follows:

- 1 Transverse colostomy provides complete decompression
- 2 It completely diverts the fecal stream
- 3 The operation is easily and safely performed by a properly trained surgeon
- 4 In summarizing the reports from the literature comparing the value of cecostomy with that of transverse colostomy it may be noted that in 85 to 95 per cent of the cases defunctionizing colostomy was considered satisfactory while this was true for only 50 to 75 per cent of the cecostomies.

Contra Indications to Transverse Colostomy

Sigmoid volvulus is an absolute contra indication to the use of defunctionizing transverse

colostomy as a decompressing procedure. This is elementary since a colostomy in the transverse colon will not decompress such closed loop obstructions as sigmoid volvulus.

Carcinoma of the colon may at times be associated with small bowel obstruction. Such obstruction is especially apt to occur with carcinoma of the sigmoid colon and is caused either by the spread of the carcinoma to the small bowel in contact with it or by an inflammatory reaction in the adherent small bowel to such a degree as to cause obstruction. Under such conditions of combined small and large bowel obstruction colostomy is of no value since the obstructed small bowel is proximal to the point in the intestinal tract at which a vent is made.

In rare instances neoplastic involvement of the mesosigmoid may so compress the inferior mesenteric artery as to obstruct it. When this occurs necrosis may occur in the pelvic colon. Cases of this type have been reported. In such cases defunctionizing colostomy is useless since necrosis and perforation of the colon as a result of loss of blood supply are not prevented by colostomy.

SURGICAL MANAGEMENT OF GASTRO- INTESTINAL OBSTRUCTIONS

Successful surgical management for the treatment of intestinal obstruction is of relatively recent origin. Praxagoras is said to have advised laparotomy in the treatment of obstinate intestinal obstruction as early as 350 B.C. Ashurst in a review of the literature in 1874 was able to find only 74 recorded cases of laparotomy for intestinal obstruction. With the passage of time however aided by the Listerian aseptic technique which made it possible to open the abdomen with relative safety and by anesthetic agents which made such surgery possible, surgeons throughout the world began to develop methods of treating intestinal obstruction by surgical means.

Early operative intervention is of the greatest importance in intestinal obstruction. If a mechanical obstruction can be relieved before distention has progressed to such a degree that interference with the blood supply to the bowel has occurred, the mortality rate should be practically negligible. The operative treatment then would vary with the lesion encountered. In any event it is important that the obstruction be relieved in some manner. At times no attempt should be made to extricate or remove the obstructing lesion because a resection might be more than the patient could survive. There is no condition which requires more gentleness than the management of acute small bowel obstruction. Bonell stated that "Every manipulation is a shove nearer the grave." There are instances in which the surgical procedure of simply draining the intestine above the obstruction might be a desirable method of treatment. Needless to say it is better to have a living patient who re-

quires a secondary operation than a dead one in whom the entire operation has been completed. With this in mind Heidenhain as early as 1897 advocated an enterostomy as treatment for acute obstruction.

The cure of acute intestinal obstruction requires the removal of the obstructing process by surgical operation. The operation may be simple—no more than the cutting of an adhesive band or as in the case of strangulating obstruction it may be complicated—involving resection of the bowel and anastomosis. There are three main objectives in the surgical approach to intestinal obstruction. These are:

- 1 Removal of the intestinal contents proximal to the point of obstruction
- 2 Relief of the distention which is paralyzing the intestinal musculature and producing physiologic disturbances of great importance
- 3 The restoration of the continuity of the intestinal tract with the re-establishment of the normal outlet for the fecal stream

In those cases in which the obstruction is external (for example incarcerated hernia) the problem is a relatively simple one. Following the relief of the obstructing process if a portion of the bowel is not viable, resection is required provided the condition of the patient will withstand this much operative trauma.

In the management of intestinal obstruction it is important to remember that surgery is being performed for a disease entity which has produced a profound disturbance in the physiology of the in-

dividual. This disturbed physiology must be corrected either prior to or at the time of surgery to insure the best possible results. In those cases in which intestinal obstruction is the result of carcinoma or some other malignant lesion, one is confronted by an additional problem, namely the management of malignancy. A malignancy, in itself, is capable of producing such vitiating effects upon its host that the surgical procedure may have to be adapted to the conditions found at the time of operation. One cannot approach the surgical management of intestinal obstruction with any preconceived ideas as to the exact surgical procedure to be performed. The specific surgical procedure indicated must be adapted to the case in question. The patient should never be adapted to the surgical procedure.

ABDOMINAL EXPLORATION

The type of incision used and its location depends upon the location and the nature of the obstructing process. A careful evaluation of the survey film and the other radiologic diagnostic studies will help immeasurably in deciding where the point of obstruction may be. In general the transverse incision is desirable. However, when no localization of the obstructing process is possible, one may resort to a right paramedian incision which can be carried upward and downward.

Once it has been opened, the abdominal cavity should be very thoroughly explored. An abnormal amount of fluid should be looked for and the character of this fluid noted. If the fluid contains bile, urine or gastro intestinal contents, then the gall bladder, urinary bladder, stomach or intestine is undoubtedly perforated. Blood stained fluid is a sign of circulatory stasis and pure blood indicates an internal hemorrhage. The venous stasis and strangulating changes in the intestinal wall are indicative of an obstruction to the mesenteric vessels. Acute pancreatitis causing obstruction is characterized by the formation of whitish or pearly white plaques noted in the fat as a result of fat necrosis. Distention of the stomach is suggestive of obstruction in the pylorus or duodenum. If one portion of the small bowel is distended and another portion collapsed, the obstruction can probably be found between them. A dilated cecum is indicative of obstruction of the colon distal to the cecum.

In order to reach the obstructing lesion it is desirable to follow the empty loops of bowel to avoid dislodging the dilated ones which are difficult to replace. When the obstruction is caused by gallstones, foreign bodies or benign pedunculated tumors, incision with removal of the obstruction and closure of the small bowel is sufficient treatment. If after dissecting the band or adhesions the surface of the bowel is denuded or the peritoneum is badly damaged it is probably better to do a primary resection of the loop involved with an end to end anastomosis. Resection of the bowel is also indicated in the treatment of an irreducible intussusception, malignant tumors and volvulus if strangulation has occurred.

Although it is desirable to carry out the abdominal exploration by starting with the collapsed bowel below the obstruction rather than by following the distended bowel down to the lesion, this preferred management is not always possible. In some cases it may be necessary to exteriorize the entire distended bowel in order to demonstrate the point of obstruction. The use of the simplified intestinal decompression sound will aid immeasurably in decompressing the entire distended bowel. This renders an adequate exploration possible in those cases in which it otherwise might be extremely difficult. By this means not only is the site of obstruction localized, but also the nature of the obstructing process can be visualized and corrected with ease. It must be remembered that the bowel may be obstructed at more than one point. Failure to find a second obstruction can have serious consequences. In any case of intestinal obstruction, particularly in intestinal obstruction due to adhesions, the entire gastro intestinal tract should be inspected for a second obstructing process.

The establishment of the viability of the involved bowel is an important decision confronting the surgeon at operation. The methods commonly used are:

- 1 Application of warm compresses to the bowel, a return of color and motility and the return of peristaltic activity and arterial pulsations to the involved segment of bowel indicate viability.
- 2 The use of 100 per cent oxygen, the fact

been suggested as a diagnostic procedure to indicate viability. If the color of the bowel definitely improves the segment of bowel will probably survive provided the administration of 100 per cent oxygen is started immediately after surgery. If the color of the bowel does not improve the segment should be resected.

- 3 Use of 2 per cent procaine by injection: the injection of 10 cc. of 2 per cent procaine into the mesentery of the suspected area will produce a release of the vasospasm. This serves as a diagnostic as well as a therapeutic procedure. In this fashion one may avoid the necessity for resection or in those cases in which resection is indicated the extent of resection may be decreased appreciably.
- 4 Injection of fluorescein: 5 to 6 cc. of 5 per cent fluorescein may be injected intravenously and the segment of involved bowel examined under ultraviolet light using an ultraviolet bulb covered with a purple glass filter. The operating room is then darkened and the intestine is observed under the ultraviolet light. If the circulation is adequate a golden green color is produced. Nonviable segments of bowel remain a deep purple color.

MANAGEMENT OF OBSTRUCTION OF THE STOMACH

The management of obstruction of the stomach depends upon the point obstructed and the obstructing mechanism as well as the condition of the patient.

Obstruction of the Cardiac Portion of the Stomach

Obstruction of Cardia Due to Carcinoma
Obstructions of the cardiac portion of the stomach due to carcinoma of the stomach with direct extension to the lower esophagus require resection of the lower esophagus and the upper portion of the stomach and re-establishment of continuity by means of esophagogastrostomy. In those instances in which the obstructing process involving the cardiac portion of the stomach proves to be non-resectable a palliative gastrostomy or the Janeway

type may have to be resorted to in order to maintain the patient's nutrition. Experience with this type of case however has led us to the conclusion that whenever possible the procedure of choice should be primary resection and anastomosis. Jejunostomy and gastrostomy should be reserved for those cases which are considered hopeless from the point of view of either a cure or a palliative resection by means of primary resection and anastomosis. The removal of the primary obstructing mechanism and the re-establishment of the continuity of the bowel by esophagogastrostomy, although not curative in many cases, do furnish a palliative procedure of great value. The removal of the primary growth permits the patient to be much more comfortable until the ultimate dissolution as a result of the carcinoma. In all these cases the pre-operative preparation is important.

The pre-operative preparation consists of correcting protein deficiencies as well as electrolyte imbalance. The former may require the use of repeated blood transfusions and tube feeding using a fine polyethylene tube passed through the esophagus into the stomach. Through this tube high protein and high vitamin liquid tube diets can be given. The electrolyte imbalance can readily be corrected by intravenous infusions. In addition to these measures large and repeated doses of broad spectrum antibiotics should be given because of the inflammatory and infected condition so commonly found in the lower esophagus. The use of an infected and edematous esophagus for anastomosis only invites leakage. By reducing such inflammatory esophageal processes to a minimum the incidence of disruption of the line of anastomosis will be appreciably reduced.

Usher has proposed a rather ingenious use of a 15 gauge polyethylene tube as a jejunostomy in those cases of non-resectable carcinoma with complete obstruction of the lower esophagus. In these cases where resection or a diversionary surgical procedure is not possible the use of a 15 gauge polyethylene tube for jejunostomy brought out through a stab wound on the side furnishes a rather simple method of feeding the patient.

Obstructive lesions of the lower end of the esophagus and upper stomach as a result of dis

orders of the esophagus are discussed in detail in Chapter 5

Obstruction of Cardia in Hiatus Hernia
The cardiac end of the stomach may be obstructed by herniation of the stomach through a diaphragmatic hernia. Although obstructions of this type can be treated by the transabdominal approach, the transthoracic approach is more suitable. Not only is the point of obstruction well visualized by this approach but the diaphragmatic hernia can be corrected at the same time.

Obstruction at the Pylorus and Lower Third of the Stomach

There are many causes for obstruction in the lower third of the stomach. These range from prolapse of hypertrophic gastric folds to pyloric cicatrizing ulcers to carcinoma of the stomach. Benign tumors are found as occasional causes of obstruction and produce a mechanical type of obstruction much like that produced by foreign bodies which are also reported as occasionally obstructing the stomach.

Carcinoma of the Antrum Obstructing the Stomach Outlet Obstruction of the lower end of the stomach by carcinoma is best treated whenever possible by subtotal gastrectomy and reestablishment of the continuity of the gastrointestinal tract by gastrojejunostomy. This may be either anterior or posterior in type. Subtotal gastrectomy for obstructing carcinoma of the antrum or lower portion of the stomach is an excellent palliative procedure even in those cases in which metastasis has already occurred. If the condition of the patient is so poor that only a minimal procedure is permissible, then a gastrojejunostomy, either anterior or posterior, may be required. In an occasional case of malignant involvement of the lower end of the stomach the carcinoma may extend up so high that a gastrojejunostomy may function poorly. Our experience with this type of diversionary procedure in the management of obstruction of the lower end of the stomach has been poor. Most patients so treated do not appear to be appreciably benefited by the relief of the obstructing process. In addition, in those cases in which the carcinoma extends well up into the stomach, the edema of the stomach

will some little distance from the obstructing carcinoma is such that the anastomosis may leak. It might be advisable in cases of this type to maintain the patient's nutrition by jejunostomy using the 15 gauge polyethylene tube. This is particularly true in those cases where the condition of the patient is so poor that only a minimal surgical procedure is permissible or in those instances when metastasis has already occurred to the liver and circumjacent tissue. Whenever possible, however, primary resection and anastomosis is the procedure of choice.

Ulcerative Lesions Obstructing the Pylorus Obstruction of the lower end of the stomach due to ulcerative lesions is best treated by subtotal gastrectomy with removal of the ulcer whenever possible. In those cases in which the condition of the patient will not permit extensive surgery or in those cases of posterior wall pyloric ulcers in which the ulcer is deeply embedded in the pancreas, a gastroenterostomy and vagotomy may be preferable. In the management of the aged patient in whom a minimal operative procedure is indicated—particularly those patients suffering from pyloric obstruction of long standing due to ulcer and associated with a low gastric acidity—a gastrojejunostomy is the procedure of choice. In general, obstructing lesions of the lower end of the stomach are best treated by subtotal gastrectomy with or without vagotomy.

Foreign Bodies or Benign Tumors Producing Pyloric Obstruction Benign tumors of the antrum may produce pyloric obstruction. These tumors may be pedunculated and obstruct the stomach by prolapsing through and obstructing the pylorus. At times the pedunculated polyp or tumor may be pushed against the pylorus by antral contractions. This acts like a cork, effectively closing the gastric outlet. A similar mechanism may occur with a prolapse of hypertrophied gastric folds.

In all cases of this type the stomach should be opened and the obstructing mechanism determined. If the obstruction is caused by a foreign body, simply removing the foreign body is sufficient. The gastrotomy opening is then closed in two layers: an inner layer of absorbable catgut and an outer one of fine silk or cotton.

Obstruction of the pylorus caused by benign

tumors prolapsed gastric mucosa and pedunculated tumors must require individualized surgical procedures. In cases of pedunculated gastric tumors obstructing the stomach outlet, simple resection of the stomach including the base of the polyp or pedunculated tumor effectively and simply corrects the problem. In cases in which there is a prolapse of the gastric mucosa through the pylorus with obstruction, a resection of the hypertrophied mucosal folds and pyloroplasty may be used successfully. There is increasing evidence, however, that even in these cases subtotal gastrectomy and gastrojejunostomy are probably the procedures of choice.

In all nonpedunculated tumors (benign) of the antrum causing obstruction, subtotal gastrectomy is the procedure which best corrects the pathology present.

Volvulus of the Stomach

In the management of obstructions of the stomach due to volvulus, the specific treatment depends upon the findings at operation. In those patients in whom the process is acute, simple detorsion of the stomach is all that is required. In those instances in which there is circulatory impairment or evidence of strangulation of some portion of the stomach, resection of that portion of the stomach with impaired circulation is required. This may require a sleeve resection in an occasional instance or it may require subtotal gastrectomy.

MANAGEMENT OF OBSTRUCTION OF THE DUODENUM

The surgical management of obstruction of the duodenum is dependent upon the etiologic mechanism producing the obstruction and the point in the duodenum at which the obstruction occurs. For purposes of treatment, the duodenum may be divided into three parts: the first limb, the descending (second portion) limb, and the transverse limb (third portion).

Obstruction of the First Limb of the Duodenum

The majority of obstructions of the first duodenal limb are due to stenosing ulcers of the duodenum. Carcinoma uncommonly causes obstruc-

tion of this portion of the duodenum. Adhesions, either acquired or congenital, may occasionally obstruct the first limb of the duodenum.

Obstruction Caused by Cicatrizing Ulcer
Those obstructions of the first limb of the duodenum resulting from ulcerations are best treated by subtotal gastrectomy and reestablishment of the continuity of the bowel by gastrojejunostomy with or without vagotomy. In those patients in whom the cephalic phase of gastric secretion is prominent, a vagotomy is essential. In the patients presenting low gastric acidity due to long-standing chronic obstruction with a greatly dilated, burned-out stomach, vagotomy may be eliminated. Generally, a vagotomy should be added to the surgical procedure in those cases of duodenal obstruction being operated upon for ulcer. By so doing, the possibility of the development of a marginal ulcer is greatly reduced.

Tumors These obstructive lesions are uncommon. Both benign tumors such as leiomyoma and malignant lesions such as carcinoma may infrequently obstruct this portion of the duodenum. The treatment depends upon whether surgery is undertaken with a view toward curing the patient or simply as a palliative procedure. At times the poor risk patient may be treated by a simple diversionary procedure because the risk of resection may be too great.

Benign Tumors The leiomyoma is usually found in the first portion of the duodenum. Obstruction from this source is uncommon, but when it does occur, it is best treated by subtotal gastrectomy and resection of the first portion of the duodenum containing the tumor. The continuity of the gastrointestinal tract is reestablished by gastrojejunostomy. In the management of the poor risk or aged patient, a diversionary gastrojejunostomy will take care of the obstruction effectively.

Carcinoma The supra papillary carcinoma is best treated by subtotal gastrectomy and duodenectomy. In those cases in which metastasis has already occurred, a palliative procedure such as gastrojejunostomy is indicated.

Adhesions Both congenital and acquired adhesions may cause obstruction of the first limb of the duodenum. The congenital cholecystoduodenocolic band has been reported as an uncommon

cause for such obstruction. Simple lysis of the adhesions effectively relieves obstruction from this source.

Foreign Bodies Ingested foreign bodies rarely come to rest in the duodenum, obstructing it. Gall stones of large size may occasionally become arrested in the first portion of the duodenum after passing through a cholecystoduodenal or choledochoduodenal fistula. Whenever possible, such foreign bodies should be milked back into the stomach and removed via a gastrotomy. If this cannot be done, the stone may be moved through the duodenum into the jejunum from whence it can be removed by enterotomy. Generally, all such foreign bodies pass through the duodenum and come to rest in the terminal ileum or successfully pass through the ileocecal valve and are excreted per rectum.

Obstruction of the Second Limb of the Duodenum

The second limb of the duodenum may be obstructed by carcinoma, duodenal diaphragm, compression by an annular pancreas, compression by tumors, or pressure from adjacent structures such as right kidney, pancreas, or hepatic flexure of the colon.

Carcinoma In those cases of carcinoma of the duodenum in which the new growth is not fixed to the circumjacent structures, primary resection and anastomosis are the procedure of choice. This is particularly true of carcinoma involving the junction of the second and third limbs of the duodenum. In the management of carcinoma involving the second limb of the duodenum, adequate treatment consists of resection of the duodenum with subtotal gastrectomy followed by gastrojejunostomy and transplantation of the common bile duct into the jejunum. In addition, the head of the pancreas must be implanted into the side of the jejunum. Unfortunately, this operative procedure is not utilized too frequently because, by the time a diagnosis of carcinoma in the second limb of the duodenum has been made, the malignancy is so extensive that a cure is not possible. The best that one can hope for in such cases is palliation which may be obtained simply by a diversionary procedure such as a gastrojejunostomy.

Duodenal Diaphragm Duodenal obstruction due to duodenal diaphragms may be satisfactorily treated by duodenotomy with cutting of the duodenal diaphragm.

Obstruction Caused by Annular Pancreas There are three main types of surgical procedure utilized in the management of obstruction of the second limb of the duodenum by an annular pancreas. These are:

- 1 Direct attack on the constricting ring. This method consists of cutting the ring and carefully ligating the pancreatic duct.
- 2 A short circuiting anastomosis around the obstruction such as duodenojejunostomy.
- 3 A combination of the first and second methods.

In some cases when a direct attack on the constricting ring has been the procedure employed and the ring is cut, there may be so much scarring of the duodenum that a functional or even a mechanical obstruction may continue. The latter may require a diversionary procedure by a gastrojejunostomy. Short-circuiting procedures around the point of obstruction have been accomplished by a gastrojejunostomy with or without gastric resection, duodenojejunostomy, or a duodenogastrostomy. Successful use of gastrojejunostomy in the management of this type of obstruction was first reported by Vidal in 1905 in the treatment of an annular pancreas associated with atresia of the duodenum. Subtotal gastric resection and gastrojejunostomy were reserved for those cases in which annular pancreas was associated with a gastric or duodenal ulcer or in which there was a huge dilatation of the duodenum, pylorus, and stomach.

Frequently, congenital anomalies may be associated with annular pancreas and should be looked for. The presence of a hypoplastic duodenum in association with annular pancreas would be sufficient indication for gastric resection and anterior gastrojejunostomy.

Gross believes that duodenojejunostomy is the procedure of choice in the surgical treatment of duodenal obstruction caused by annular pancreas, since it completely relieves the duodenal obstruction and does not interfere in any way with gastric function. All surgeons are aware of the danger of cutting the pancreatic ring with the attendant

possibility of pancreatic fistula. Gastroenterostomy alone or subtotal gastrectomy has given good results in the cases reported.

Obstruction of the Third Limb of the Duodenum

This portion of the duodenum is commonly obstructed by carcinoma duodenal diaphragm or arterio-mesenteric vascular compression and rarely by pressure from adjacent organs or structures.

Carcinoma In those cases of carcinoma of the third limb of the duodenum in which the new growth is not fixed the procedure of choice is a primary resection and anastomosis. Once the superior mesenteric vessels are isolated, identified and retracted out of harm's way the carcinomatous area and the duodenojejunal flexure may be resected with ease and an anastomosis made end to end between the duodenum and jejunum. It was formerly believed that an end to end anastomosis between the jejunum and the duodenum was rather hazardous because the posterior wall of the duodenum was devoid of peritoneum. Recent experience has demonstrated that anastomosis between the duodenum and jejunum can be performed with a high degree of safety. In those cases in which the new growth is more extensive and invades the pancreas the only chance for a complete removal would be the performance of a pancreaticoduodenal resection and end to end anastomosis between the jejunum and proximal duodenum. However in those cases in which the superior mesenteric artery and vein are involved resection is impossible because the blood supply of the entire small bowel would be compromised. Duodenal obstructions in the third limb of the duodenum to the left of the mesenteric artery and vein may be treated by duodenojejunostomy in those instances in which primary resection and anastomosis are not possible.

Duodenal Diaphragm This type of duodenal obstruction is best treated by duodenotomy and resection of the obstructing diaphragm. When this is not possible a diversionary gastroenterostomy or duodenojejunostomy may suffice.

Arterio-mesenteric Vascular Compression When obstruction of the duodenum is caused by pressure of the superior mesenteric vessels the

procedure of choice is posterior gastrojejunostomy as a diversionary procedure. No attempt should be made to relieve the obstructing process.

MANAGEMENT OF SMALL BOWEL OBSTRUCTION

The management of acute small bowel obstruction may involve a relatively simple procedure such as lysis of one adhesive band or it may involve the performance of complicated surgery with resection of a considerable portion of the small bowel. Good anesthesia is of inestimable importance during the performance of surgery for obstruction of the small bowel. No surgery on the obstructed small bowel should be undertaken in the presence of intestinal distention without having a long intestinal decompression tube down in the gastro intestinal tract or at least in the stomach. The surgeon must use his own judgement in determining the exact type of operation and the amount of exploration which should be performed in each individual case. The extremely sick patient requires only the simplest possible procedure particularly if peritonitis is present. Widespread separation of recent inflammatory adhesions may cause denudation or tears in the friable and inflamed bowel. In addition such separation may spread the existing peritoneal infection.

From the point of view of surgical management all cases of small bowel obstruction may be divided into two large groups: (1) those obstructions which are nonstrangulating and (2) all strangulating obstructions. The surgical management differs in each of these groups.

Nonstrangulating Obstruction

In nonstrangulating obstructions of the small bowel the operative procedure may be simple. In those cases in which an adhesive band is the source of the obstruction if the surgery is undertaken early so that distention of the small bowel is minimal lysis of the obstructing band will quickly result in a cure. This also applies to the management of all nonstrangulating small bowel obstructions operated upon before distention becomes marked and the patient develops electrolyte imbalance and dehydration phenomenon.

The management of nonstrangulating obstruc-

tion operated upon relatively late in the course of the disease when distention is prominent may require complicated operative procedures. The distended bowel may make recognition of the pathologic changes and intra abdominal manipulation very difficult. In addition the friability of the intestine due to the intestinal distention may be such that only the gentlest handling is permitted in order to prevent the bowel from breaking.

Although it is accepted surgical theory that the less manipulation there is the higher is the percentage of recovery, evisceration may be required in such cases. One should not hesitate to eviscerate in those cases in which visualization and accessibility of the site of obstruction are required. A failure to do so may result in the definite danger of overlooking a strangulated loop of bowel. In such cases with marked intestinal distention the distended bowel should be emptied of its contents before the obstruction is released. This may also be necessary in those instances in which the obstruction is released without emptying the distended bowel because the return of such distended bowel to the peritoneal cavity is no simple task. By emptying the distended bowel after surgery the collapsed bowel can be returned to the peritoneal cavity relatively easily and the abdomen closed. To obtain such emptying of the distended bowel the Cantor decompression sound may be found to be useful. This should be resorted to only in those cases in which the long intestinal decompression tube has not passed down the gastro-intestinal tract successfully. If the tube has passed beyond the ligament of Treitz it is a relatively simple matter to rapidly thread the intestinal tube down to the point of obstruction thus decompressing the bowel. A complete discussion of this technique is presented in Chapter 18. Our experience as well as that of many surgeons throughout the country has demonstrated that the aseptic deflation of the distended bowel at the time of surgery is a safe and practical procedure. The fact that the mortality rate has been lowered in those cases in which this method was used establishes its value rather conclusively.

Strangulating Obstruction

One of the worst conditions that the surgeon is called upon to treat is strangulation of the bowel

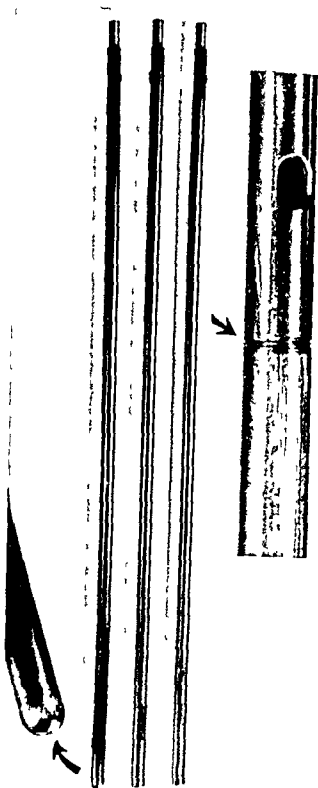


FIG. 36. Cantor intestinal decompression sound.



FIG 367 Note the end of the Cantor intestinal decompression sound which is rounded to avoid injury to the mucosa. There are three spirally placed holes of 20 Fr diameter.

since one can never be certain of the exact extent of the disease and must rely upon gross changes which may not be as accurate as one would desire. Generally in such cases it is better to resect the small bowel at a point high enough to insure an adequate blood supply.

At the present time one may say with a reasonable degree of assurance that with the use of antibiotics little or no danger to the patient will result from peritoneal soiling at the time of surgery if the anastomosis is carefully performed, the intestinal distention treated and the bowel kept empty. Open methods of end to end anastomosis and intestinal decompression at the time of surgery can be performed meticulously without the



FIG 368 Note the point of union of the segments of the Cantor decompression sound. These segments are smooth and do not injure mucosa. The hole at the end of each segment is a make or break hole. With the index finger over the hole suction is obtained by raising the finger; suction is broken.

necessity of closed resection. Recent experience has established the fact that contamination at the time of surgery is not lethal. Rather it is the continuous leakage of intestinal contents caused by a leak at the site of anastomosis or a leak at the site of necrotic areas in the bowel with continuous spillage that causes death. In our experience we have noted on several occasions that 200 to 300



FIG. 369 Marked intestinal distention is noted upon opening the abdomen of a patient with small bowel obstruction not previously decompressed with the intestinal decompression tube. Note the difficulty with which the distended loop of bowel are held within the abdomen.



FIG. 370 Illustration of the technique of plicating small bowel and decompressing it by threading upon a Cantor decompression sound. The technique and use of this sound is simple.

cc of intestinal contents have been poured into the peritoneal cavity without harmful effects to the patient. All that was required was that the peritoneal cavity be thoroughly flushed out with normal saline, as much of the intestinal contents aspirated by suction as possible, the intestinal de-



FIG. 371 Marked small bowel distention associated with intestinal obstruction of the small bowel. The patient presented characteristic signs of impending strangulation. Immediate surgery was indicated despite intestinal distention.

compression tube passed successfully into the small bowel, the rent in the bowel properly repaired, and the patient markedly treated with antibiotics. This almost invariably produced a successful outcome.

Various restitutive measures may be used in the management of strangulated loops of bowel. Warm packs may be used instead of the hot packs which damage the serosa of the bowel. The following measures have been reported as being of value as additional procedures:

1. Oxygen inhalation should be used along with vasodilating drugs both to increase the oxygen supply to the blood vessel and to demonstrate the point at which there is impairment of the blood supply to the small bowel. In that portion of the small bowel in which the blood supply is adequate a definite pinking of the bowel occurs, whereas in that por-

tion of the small bowel deprived of its blood supply no such change takes place

- 2 Injections of 5 to 10 cc of 2 per cent procaine hydrochloride into the mesentery of the bowel adjacent to the loop affected have been shown to be of value as a therapeutic as well as a diagnostic measure. In many of the cases reported as well as in experimental animals immediate improvement occurs.
- 3 Although heparin has been used to prevent further propagation of a thrombus, Laufman among others has warned against its use. Laufman has shown experimentally that heparin increases the danger of hemorrhage into the lumen of the bowel to such an extent that animals so treated may bleed to death. Heparin however is useful in preventing the propagation of a thrombus after resection of an infarcted segment.
- 4 Papaverine has been found to be of some value in relieving residual vasospasm in the released strangulated loops in animals. Laufman and Method demonstrated experimentally by means of surface temperature readings that there was considerable residual vasospasm in the minute vessels of a still viable intestinal loop. Because of the factors which affect the recoverability of a strangulated loop of bowel this vasospasm is of no small importance whether the strangulation is primarily arterial or venous. Release of a viable arterial type of strangulation results in a reactive hyperemia followed by a phase of vasospasm. Release of a viable venous type of strangulation is followed by a slow rise in temperature up to or almost up to the control level. In their experimental studies Laufman and Method found that papaverine hydrochloride was of value in releasing this residual vasospasm which provided the loop was recoverable aided the recovery of the bowel following strangulation. Comparative studies of the effects of warm packs, oxygen inhalations, injections of novocaine into the mesentery of the bowel and intravenous administration of papaverine demonstrated that these measures all had some value in increasing the blood flow

through a still viable loop of intestine. When papaverine in large doses was given following one or a combination of these resuscitative measures an additional response was rapidly seen.

Among the tests for viability of the bowel the following may be found to be of some value:

- 1 A return of pulsations to the vessels supplying the segment of bowel involved. This may be confusing, at times since pulsations may be transmitted through thrombotic vessels in a loop of bowel in which irreversible damage has occurred.
- 2 The return of normal color to the bowel—either a pink or red color. This is extremely important. The persistence of a dusky color with engorgement of the mesenteric veins suggests loss of viability.
- 3 The return of peristaltic activity. This may not be a good prognostic sign since the bowel may go into spasm at a later date as a result of anoxia.
- 4 The injection of fluorescein. For this method refer to the first portion of this chapter.
- 5 An increase in temperature of the segment of bowel after release of the obstruction has been described as a reliable test for viability.

A review of the current literature as well as our own personal experience has demonstrated that there is actually no single foolproof test to tell us whether any given loop of bowel is completely viable. The absence of mesenteric pulsations, the absence of peristaltic contractions, the failure of a loop of bowel to regain a good pink color, rather quickly increased thickness of the bowel wall with edema and hemorrhage of the mesentery are all indications for resection. A completely black loop of bowel with a fetid odor is the end result of extravasation of blood into the lumen as well as into the wall of the bowel. This may be due to hypoxia followed by capillary damage. Although the color in such a loop of bowel may return upon the release of the strangulating mechanism the mucosa has been damaged irreversibly. Clinical experience has demonstrated that 20 to 30 minutes are usually the extreme limit during which a questionable viable loop of bowel should be observed.

It must be pointed out however, that in an occasional case in which a loop of bowel of questionable viability is returned to the peritoneal cavity postoperative fibrotic changes may occur causing a fibrous stricture of the small bowel. This may cause chronic intestinal obstruction as a result of stenosis. These changes of stenosis and fibrosis may occur in a loop of small bowel which was considered to be viable at the time of the initial operation. The latter change is most likely to occur as a result of strangulation of a loop of bowel in a hernial sac.

PARALYTIC ILEUS

In the management of paralytic ileus surgical intervention is definitely contra indicated except to treat the causative factor (for example the drainage of a pelvic abscess). This type of obstruction is best treated by conservative means using the long intestinal decompression tube. The methods used are presented in Chapter 19.

ENTEROSTOMY

This subject is presented in detail in Chapter 20. At the present time there are very few indications for its use.

BOWEL RESECTION

Intestinal resection with end-to-end anastomosis has more or less been considered the procedure of choice whenever resection of strangulated bowel is required. In those cases of intestinal obstruction in which removal of the obstructing process is technically impossible due to the poor condition of the patient a diversionary entero-enterostomy around the obstructing process may often be a lifesaving procedure.

Despite the essential role which the small bowel plays in digestion it has been possible to remove up to 8 feet without causing serious disturbances in the digestive function. Ribas reports the case of a 13 year-old child in whom almost the entire jejunum and ileum were removed. Although the youngster gained weight and apparently was clinically well under hospital management upon discharge to her home she could not be kept in a reasonable state of nutrition and as a result of the disturbances in intestinal physiology survived only five months.

At times extensive lengths of small bowel may require removal. The term extensive is applied to resection of the small intestine is generally understood to include only those lengths measuring 6 feet or more. This measurement is accepted on the basis of records which indicate that resection of the bowel beyond 6 feet may give rise to various metabolic disturbances.

Length of Small Bowel Normally Present in Man

The length of small bowel normally present in man has been found to vary. Some authors put the length of the small intestine between 15 feet 6 inches and 31 feet 10 inches. Sir Henage Ogilvie noted that in the African native much longer lengths of small bowel have been reported. The general average of small bowel would be approximately 22 feet 6 inches. Bremner reported that the length of the small bowel varied widely in direct ratio with the height and weight of the individual—the larger and taller the individual the longer the small intestine. Beneke stated that for every 3 feet $3\frac{1}{2}$ inches of body length there were 12 feet 9 inches of small bowel. Hunt believed that there was a definite ratio between the size of the individual and the epithelial surface of the intestine.

How Much Small Bowel Can be Removed

In performing a resection of the small bowel for intestinal obstruction with strangulation of the bowel the problem arises as to how much small bowel may be removed without seriously compromising the nutritional status of the patient. The important question in deciding how much bowel one can resect with safety is not how much bowel is to be removed but how much will be left behind. Doerflinger reported a patient in apparent good health six years after removal of 18 feet 8 inches of small bowel. Only 44½ inches of jejunum and 8 inches of terminal ileum were left. Because the patient was in apparent good health had no complaints and ate regular meals Doerflinger questioned whether the small bowel was really essential for life. Bremner in a review of the literature up to 1928 found approximately 83 cases reported (with 71 recoveries) in which more than 6 feet

7 inches of small intestine had been removed. The conclusions which may be drawn from a review of the literature are essentially that the minimum resection may fail at times and that the maximum resection may sometimes offer excellent results but that the answer lies somewhere between the two extremes. An arbitrary safety limit of 6 feet 7 inches when applied to a resection of the small bowel has been established. Resections up to and beyond this arbitrary limit however may become necessary as lifesaving measures and have yielded an 85 per cent recovery from the operation with a 65 per cent recovery of function.

It is a well known fact that in some cases of chronic inflammatory disease of the small bowel marked shortening of the small bowel may occur. In cases of this type a resection of 6 feet 7 inches might represent 75 per cent of the whole small bowel. The consensus at the present time appears to be that the removal of more than three fourths of the small intestine regardless of its total length is likely to have serious permanent effects. Since there is considerable variation in the length of the small bowel in different individuals the total amount of small bowel which may be removed is variable. It is generally agreed that up to half of the small bowel can be resected without fear of metabolic disturbances. If the circumstances make it impossible to estimate the exact length of bowel then 6 feet 7 inches must be considered as the ultimate length of bowel which may be removed satisfactorily.

Except in those unusual instances in which more than 75 per cent of the small bowel is removed extensive resections often give better results than one would expect. Consequently within limits the surgeon should remove that amount of bowel which his judgement tells him is adequate. One of the most extensive resections of the small bowel was that reported by Jackson and Linder. In this patient a 27 year old man the entire small bowel except for 7 inches was resected. After recovery from this operation the patient returned to work for six months. At the end of this time he developed severe pellagra. Abnormalities of behavior with epileptic seizures and finally true pellagic psychosis of the mixed type developed and failed to respond to vitamin therapy. Although

many patients with massive resections do develop diarrhea this specific patient did not and the colon showed no evidence of colitis. Gastrointestinal studies showed a hypochlorhydria. There was a marked decrease in the pancreatic and intestinal enzymes and the pH was unfavorable for their action. Observation of intestinal absorption in this patient revealed a large loss of protein phosphate calcium and salt in the stools with almost complete absence of fat absorption. In addition there was a deficiency in the absorption of vitamin A, B and C. The patient usually had one bowel movement a day which was pale and without fecal odor and contained only slight traces of indol and scatol. Water and salt were absorbed and temporarily retained in the body. They were excreted after a short interval usually at night. The patient was still alive two years following surgery although he had developed all the characteristics of pellagra.

In those cases in which massive small bowel resection has been performed in addition to the metabolic disturbances discussed tetany should be watched for as a possible postoperative complication.

Compensatory Mechanism of Recovery After Extensive Resection

It has been suggested that functional recoveries in man as well as experimental animals are dependent upon a compensatory hypertrophy of the small bowel. The fact that functional recovery depends upon a compensatory hypertrophy of the remaining small bowel has been well demonstrated in experimental animals in which there is an increase in the transverse diameter of the remaining small bowel. There is not however an increase in villi in this enlarged bowel. Such compensatory hypertrophy has not as yet been reported in humans although some such mechanism is postulated.

In many patients in whom long loops of small bowel equal to or slightly beyond the arbitrary limit are excised severe metabolic disturbances appear relatively early. Clinical studies in experimental animals demonstrated an excess of nitrogen and fat in the stools as a direct result of a diminution of the absorbing surface of the body. Profuse diarrhea generally occurs after the operation and

a loss of weight becomes pronounced. After a variable period of time the excessive excretion of nitrogen and fats decreases. When this occurs the animals return to a normal state of nutrition. They remain susceptible to changes in diet, however, so that diets rich in proteins, fats or indigestible substances may again produce diarrhea. Similar conditions occur in humans subjected to such extensive surgical procedures on the small bowel. This pattern of recovery suggests the development of a compensatory increase in the mucosal small bowel absorbing surface which can only be brought about by an increase in the diameter of the lumen of the residual bowel.

Operative Procedures

The operative procedures that may be used in resecting bowel are the side to side anastomosis, the end to end anastomosis and the end to side anastomosis. Of these three procedures the end to end is rapidly becoming the procedure of choice for all portions of the small bowel. In recent years, however, Trice and Giles have pointed out that in the terminal ileum end to side (end of ileum to side of colon) is a better procedure.

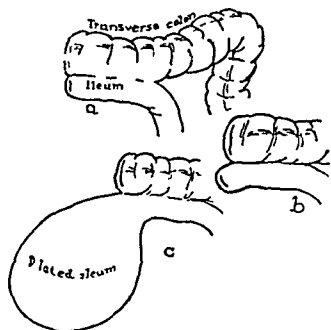


FIG. 37. Markedly redundant blind segment of intestine following side-to-side anastomosis. Ulcerative and inflammatory changes occur in this dilated segment causing diarrhea.

Side to Side Anastomosis For many years side to side anastomosis was widely used in bowel resection. With the passage of time it was found that this type of anastomosis presented certain disadvantages. One of the most important of these is the development of the blind loop syndrome. This syndrome is apt to occur if an excessive length of the proximal bowel is permitted to remain distal to the anastomosis. When peristaltic activity carries the intestinal contents toward the blind end, the pouch may dilate three or four times its normal diameter. The pouch formed in this fashion then may retain feces. Ulceration can occur and perforation is not uncommon. On the other hand, a blind loop becomes a functional segment. If peristaltic activity moves in the direction of the anastomosis, this redundant segment may give rise to a blind loop syndrome. Hypertrophy and elongation of blind loops of ileum have been observed at autopsy years after side to side anastomosis of the colon and small bowel. Attacks of colicky pain, vomiting, and diarrhea are not uncommon with this blind loop syndrome. Ulceration in itself does not appear to produce definite symptoms and often the first evidence of this condition is peritonitis due to perforation. In general, it may be said that considerable time may elapse between the bowel resection and the onset of symptoms of blind loop syndrome.

It has been suggested that one of the reasons that side-to-side anastomosis results at times in leakage is the fact that the opening between the two loops of bowel is placed at a point between the mesenteric and antimesenteric borders of the small bowel. An opening placed at this point may interrupt the blood supply to the bowel edge on the antimesenteric side of the anastomosis. The studies of Cokkinis suggest that this is possibly the explanation. He demonstrated by injection studies on human bowel that collateral circulation in the small bowel stops where the terminal row of vascular arcade approaches the antimesenteric border of the bowel. As a result, the antimesenteric portion of the small bowel presents a meager blood supply.

End to End Anastomosis The advantages of end to end anastomosis after bowel resection are that it is simple, anatomically feasible, and in

involves the least amount of suturing. The trend at present is toward end to end anastomosis.

The following technique has been found useful in anastomosis, end to end two loops of bowel of different luminal diameters. The loops of bowel to be anastomosed are placed side by side with the mesenteric edges together and the antimesenteric edges together. The first row of nonabsorbable interrupted sutures (chromic) begins at the mesenteric edge and proceeds to the antimesenteric

edge. When the edge of the smaller bowel (antimesenteric) is reached the interrupted sutures are continued along the longitudinal axis of the antimesenteric edge of the smaller bowel. These sutures are inserted until the entire lumen of the larger bowel has been sutured to the smaller bowel. The lumen of the smaller bowel is then increased in size by cutting along the antimesenteric edge of the small bowel just above the row of chromic sutures. The inner row of absorbable sutures is then inserted in the usual fashion. With this technique it is possible to anastomose end to end loops of bowel of widely varying luminal diameter.

End to Side Anastomosis This type of anastomosis is most useful in resections of the small bowel at the terminal ileum. In this region of the terminal ileum there are very serious disadvantages to an end to end anastomosis. Among these are the following:

- 1 The blood supply in the terminal ileum may not be adequate.
- 2 The back pressure of the ileocecal valve may cause a leak in an end to end anastomosis in close proximity to the valve.
- 3 It may not be safe to perform an end to end anastomosis in this area because of the ileocecal fat pad and the appendix. The bowel edges to be anastomosed anywhere in the gastrointestinal tract should be free of fat and adventitious tissue. In the terminal ileum this may be possible.

For all these reasons it has been suggested by many surgeons that ileocolostomy with closure of the distal end of the ileum is the procedure of choice in lesions requiring resection of this portion of the gastrointestinal tract. The Cook County Hospital review of 15 cases with small bowel obstruction which required an anastomosis about the terminal ileum would seem to bear out the advantages of end to side anastomosis in this area. This review pointed out that 30 per cent of all the end to end anastomoses performed in this small series developed a leak at the line of anastomosis whereas none of the end to side anastomoses did so. Although these figures do not involve large enough numbers to really constitute an absolute proof yet they do suggest that unless operating conditions are optimum with a good

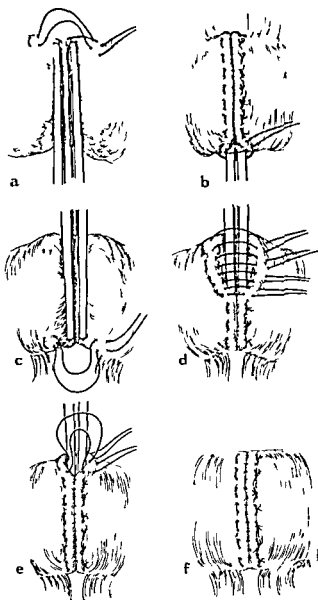


FIG. 373. Dennis technique of closed end to end anastomosis over clamps.

blood supply in the terminal ileum and no interference to the bowel wall from adventitious tissue or edema; an end-to-end anastomosis should not be performed in this area. Trice and Giles consider an end to side ileocolostomy with closure of the distal portion of the ileum a much safer operation.

MANAGEMENT OF SMALL BOWEL CARCINOMA

It was formerly believed that primary malignancy of the small bowel did its greatest damage because of mechanical obstruction and not because of metastasis. Time however has proved that the reverse is true. The present concept is that radical resection with re-establishment of the continuity of the bowel is the treatment of choice in such cases. Experience has shown that in the treatment of carcinoma at times even a resection of the small bowel which appeared to be inadequate based upon anatomic and pathologic lines has been followed by years of health without recurrence of the malignancy.

If radical resection is not possible diversionary procedures above and below the obstructing lesion should be performed to give relief from the obstructive process. Stricture or obstruction of the bowel in most cases occurs long before the effects of regional or remote metastasis have manifested themselves. As a result if the surgeon is aware of the possibility of carcinoma and correctly diagnoses it wide resection of the tumor and the gland bearing areas tends to give the best results. Most cases of carcinoma of the small bowel spread by direct extension to neighboring viscera and the regional lymph nodes. Rarely is a growth disseminated to distant organs even in the terminal phase. For this reason and because the carcinoma tends to remain localized to the small bowel for long periods of time even in the presence of lymph node metastasis primary resection and end-to-end anastomosis give very satisfactory results.

MANAGEMENT OF LARGE OBSTRUCTED HERNIA

At the 44th annual meeting of the Western Surgical Association in 1926 Bartlett called attention to the management of intestinal obstruction resulting from incarceration in a large ventral or

umbilical hernia in the obese individual. He noted that following surgery the mortality rate in these individuals was quite high since they suffered from an increased intra abdominal pressure upon reduction of the obstructed hernia. One further increase in the intra abdominal pressure when one closes very large umbilical or ventral hernial defects without first reducing the weight of the patient to approximately the average weight. For those patients in whom one is able to reduce the hernia and close the defect under very great tension a characteristic train of symptoms has been reported to develop almost immediately because of the decreased vital capacity due to the increased intra abdominal tension.

The following symptom complex results from such increased intra abdominal tension in the obese. The patients become flushed, their pulse rate rises rapidly, their blood pressure goes up and breathing becomes dyspneic. Pulmonary edema and atelectasis may develop within the first 24 hours. These patients may take deep breaths in the course of their struggles and cough or vomit. In some instances the patients are immediately relieved as a result of a wound dehiscence. When this occurs the breathing becomes more comfortable, the pulse rate drops, the temperature will fall and the blood pressure returns to the pre-operative levels. This is a sign that the entire posterior suture line has pulled out. If the patients do not get this relief they may die. The mortality rate is approximately 25 per cent in such cases.

Therefore in obese individuals who have large hernial defects with incarceration it is best to open the sac in place of the contents and if no circulatory interference has taken place merely cut the neck of the sac. This increases the opening and prevents the development of strangulation. The abdominal wall may then be closed with no attempt made to repair the large hernial defect unless this can be done without undue tension. The hernial repair is then postponed for a later date when the patient has lost weight and is in better condition to be subjected to an increased intra abdominal pressure. The recommendations by Bartlett have not been widely accepted but nevertheless offer a conservative approach to the problem.

We have rarely found this condition require

necessary. Very large hernial defects can readily be repaired at the time of surgery if the patient is well relaxed and well aerated by means of endotracheal anesthesia and if the dissection is carried back far enough so that the tissues may be brought together without markedly increasing the tension. In addition in order to decrease the amount of tissue which one puts back into the abdomen, all herniated fatty omentum which almost invariably is found to occupy the hernial sac, should be removed and not replaced in the peritoneal cavity. Early ambulation of such patients and decompression of the upper gastrointestinal tract by means of a long intestinal decompression tube have made it unnecessary for us to leave such hernial repairs for a later date.

ENTERO ENTEROSTOMY

When intestinal obstruction is caused by a mitting together of several loops of bowel with no evidence of strangulation and the condition of the patient does not permit extensive dissection or bowel resection, the operation of choice may be a diversionsary anastomosis around the obstructed and adherent bowel. Trendelenberg first proposed this operative procedure in 1885. Shortly after in 1888 von Hacker also described this principle of intestinal exclusion. Salzer first used this procedure for lesions of the cecum in 1891 and a modification of the procedure is familiar today in the form of an end-to-side ileocolostomy with exclusion of the cecum. The purpose of this type of exclusion operation in intestinal obstruction is to restore the continuity of the intestinal tract and to by pass the point of obstruction.

Estes and Holm reported in 1932 that in clinical cases as well as in experimental animals an ulcerative enteritis may develop in these by passed obstructed loops of bowel. When this occurs the following syndrome appears. The patient begins to vomit and have diarrhea at the same time. Overactive peristalsis and loud borborygmus are noted on abdominal auscultation. On examination the involved intestinal loops are found to be dilated.

In 1937 Estes reported five cases in which an entero anastomosis had been performed to relieve an intestinal obstruction due to massive adhesions. In these cases an enteritis of the obstructed loops

developed as indicated by recurrent attacks of diarrhea, abdominal pain, overactive peristalsis and at times vomiting, all of which were entirely relieved by resection of the side tracked loops of bowel. In the e loops there was apparent stasis as a result of some persistence of the obstruction or constriction of the bowel. This caused enteritis and ulcerative mucosal changes. The more complete the obstruction was the more evident the ulcerative process became. Estes demonstrated experimentally in dogs that with the performance of an anastomosis around an intestinal obstruction whether an ileo ileostomy or an ileocolostomy, the closed loop syndrome was apt to occur. In addition in the experimental animal the enteritis was found to extend into the ileum proximal to as well as into the colon distal to the point of obstruction.

Pearse demonstrated in experimental animals that short jejunal loops up to a certain length were well tolerated and emptied themselves spontaneously. Loops of small bowel from 3 to 4 feet long became inspissated with fecal material and debris and dilated enormously. In these cases if perforation did not occur emaciation, anorexia, lassitude and finally death might follow. Loops of bowel 5 to 6 feet long could cause death from inanition and dehydration. Pearse described a clinical case in which six months after a lateral anastomosis around an obstruction the patient developed attacks of abdominal discomfort, anorexia, nausea, vomiting and loss of weight. Resection of the side tracked segment was followed by diarrhea for three weeks after which the patient remained well. The side tracked segment of bowel was found to be thick, dilated and markedly ulcerated.

Urmy, Ragle, Allen and Jones reported a case of beriberi which followed a short circuiting of the small intestine around an obstructive process.

Cockett called attention to the fact that there have been well over 300 cases reported in the world literature in which massive resection of the small intestine has been successfully carried out without the patient showing marked degrees of starvation. The only residual symptom in most of the cases that remained alive was a mild to moderate fatty diarrhea which was controllable by limiting the dietary fat. For this reason Cockett sug-



FIG 374 Notice emaciation of patient and marked edema as a result of hypoproteinemia due to the intrac table diarrhea resulting from jejunal stricture and the blind loop syndrome

gested that the symptoms of ulcerative enteritis as a result of entero enterostomy were not due to the fact that the gastro intestinal tract was deprived of a large segment of bowel utilized for nutrition but rather were due to the fact that such operations leave a large closed loop of small bowel. These closed loops become distended and since distention is the physiologic stimulus for intestinal peristalsis hyperperistalsis very commonly occurs. As a consequence of this hyperperistalsis food and nutrient material taken by mouth pass through the gastro intestinal tract so rapidly because of the powerful irritable focus exerted by the distended isolated loop that the patient literally starves to death. All such patients show marked nutritional deficiency with loss of weight diarrhea and occasionally pain. Severe hypoproteinemia is extremely common in these patients.

EXTERIORIZATION PROCEDURE IN SMALL BOWEL OBSTRUCTION

The basic principle of exteriorization can be directly applied to the surgical management of strangulating obstruction of the small bowel in which the condition of the patient is such that primary resection and anastomosis which is the

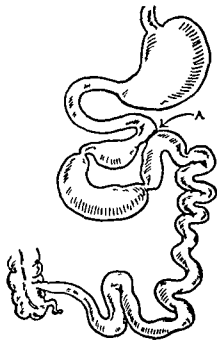


FIG 375 Condition found at surgery in patient in Figure 374. Note the intestinal distention in the isolated loop. Ulcerative and inflammatory changes occurred causing intractable diarrhea.

procedure of choice cannot be performed with safety. The only prerequisite for exteriorization is a sufficiently mobile bowel. In those cases in which the method can be applied the immediate advantages are as follows:

- 1 The maneuver is not time consuming.
- 2 It may be done rapidly and simply.
- 3 The involved segment of bowel may be brought outside of the abdominal cavity and in this way one avoids the possibility of peritoneal contamination.
- 4 The abdominal distention can be brought under control within a short period of time by securing an outlet for the intestinal gases and content by an opening in the proximal limb.

It should be emphasized however that such exteriorization procedures need rarely be done. Whenever possible primary resection and anastomosis is the procedure of choice in all cases. In some isolated instances however or those cases seen relatively late in the course of disease in which the condition of the patient is too poor to permit adequate surgery such exteriorization procedures may be lifesaving.

The method of exteriorization which can be utilized in the management of poor risk strangulating types of obstruction from whatever cause cannot be applied in those cases in which there is not sufficient mobility of an involved segment to permit exteriorization. In addition if the mesentery is edematous and well fixed the bowel may not be lifted and freed from its position without additional dissection which would be a rather time consuming procedure and defeats the purpose of this operative procedure. The most serious disadvantage in this procedure occurs in childhood and is due to the extensive loss of intestinal fluid through the double barrelled enterostomy. This aggravates an already disturbed electrolyte balance and the nutritional status of the patient suffers. As a result such exteriorization procedures should rarely if ever be performed on infants and children. To offset this disadvantage Woodhall and Cokkinis recommend a side to side anastomosis as an additional step in the operation of exteriorization of the small intestine for acute intestinal obstruction with gangrene. The side to side anastomosis is made 10 to 12 cm. from the exteriorized end. The terminal ends are temporarily closed by clamps. By intermittently opening the clamp on the small intestinal limb abdominal distention can be immediately counteracted while the proximally placed entero anastomosis results in a restoration of the continuity of the bowel. Closure of the enteric openings in the abdominal wall is then carried out in 10 to 17 days following the emergency surgical procedure. The chief usefulness of this operative procedure occurs in those cases in which strangulated or gangrenous bowel is found and in which the condition of the patient is such that complete resection and primary anastomosis could not be tolerated.

If the side to side anastomosis has been performed at the time of exteriorization then the closure of the enteric openings in the abdominal wall is feasible by invaginating the distal stumps of the intestine and returning them to the peritoneal cavity. If a side to side anastomosis has not been performed the enteric limbs should be resected and an end to end anastomosis made in healthy bowel.

MANAGEMENT OF IRREDUCIBLE INTUSSUSCEPTION

A small number of intussusceptions are irreducible. In such cases a number of operative procedures have been devised in order to correct the situation. The operative procedures devised depend upon both the condition of the bowel and the condition of the patient at the time of surgery. Two types of operation which were formerly used are now rarely necessary in this era of good anesthesia, adequate blood and antibiotics. These procedures however may be useful in an occasional case and are discussed for the sake of completeness.

Jejunostomy Ileostomy or Colostomy

Any of these surgical procedures might be used as an initial operation in a seriously ill patient in the hope of saving him for the time being by decompressing the intestinal tract proximal to the point of obstruction. In those cases in which this primary procedure is successful it should then be followed by one of the other definitive surgical procedures for the treatment of the obstructive intussusception.

Entero anastomosis

This operation consists of short-circuiting the bowel around the obstructing intussusception. It too has been used as a first stage decompression procedure to be followed later by secondary removal of the intussuscepted bowel. There are some surgeons who have used this procedure as a complete operation in itself leaving the incarcerated gangrenous segment of bowel to slough off eventually and thus pass down the gastro intestinal tract for excretion per rectum. It is hoped in cases of this type that the constricted area at the neck of the intussusception would by that time be sufficiently surrounded by inflammatory reaction to prevent leakage.

However one of the more serious drawbacks to this type of operation is the failure of the sloughed portion of bowel to be excreted per rectum. A gangrenous bowel may not pass spontaneously through the gastro-intestinal tract and intestinal impaction with obstruction may result. Such a case was reported by Mayo in which the acute obstruction

finally released itself with the passage per rectum of a segment of gangrenous bowel 4 feet long

There are two recognized procedures which are useful as emergency treatment in cases of irreducible strangulated intussusception. These are

- 1 Exteriorization of the gangrenous irreducible portion of the gastrointestinal tract. This exteriorized bowel is then cut away leaving two enterostomy openings. These are closed at a later date.
- 2 An intussusception type of small bowel anastomosis has been proposed as a rapid safe procedure for the poor risk patient. The method consists essentially in the insertion of sutures tacking the intussusceptum to the intussusciens at its neck, opening the intussusciens, resecting the intussusceptum and with one row of sutures, anastomosing the mucosal surfaces of the intussusciens to the intussusceptum and then closing the opening in the roof of the intussusciens. Experimental and autopsy studies have demonstrated that smooth anastomosis results from this technique.

Whenever possible intestinal resection with primary anastomosis is considered the procedure of choice in the management of irreducible intussusception. The recent report of Dennis on eight intestinal resections with 100 per cent recovery indicates the trend toward surgery in the management of this type of case particularly in infancy and childhood. In such small patients a closed anastomosis with immediate restoration of intestinal continuity is highly desirable to prevent loss of intestinal contents and disturbances in electrolyte balance. In addition in all patients with gangrenous and irreducible intussusception the obstructing segment with the incarcerated gangrenous portion of the bowel is removed *in toto* and the remaining bowel which is viable and healthy is primarily anastomosed.

In the performance of primary resection and anastomosis Ladd and Cross reported that in 28 per cent of the cases the most advanced portion of the intussusceptum could be felt by careful rectal examination. Because of this if one were to resect the entire intussuscepted portion of the bowel most of the gastrointestinal tract might require

removal. However when one considers that the most distally advanced intussusception does not necessarily prove to be irreducible such dangers may be more theoretical than real. In the management of cases with a long intussusceptum which proves nonreducible if the case lends itself to intestinal resection, an advantageous maneuver might be to manipulate the invaginated segment to a proximal position. By so doing it is possible to coil up the invaginated segment into a ball immediately distal to the neck of the intussusception at which point the resection can be carried out. In this way one would tend to minimize one of the drawbacks of primary resection and anastomosis in irreducible intussusception i.e. the necessity of including in the resection that portion of the bowel which is representing the enveloping sheath and which is potentially normal bowel. In addition one may reduce to some degree the extent of the bowel to be sacrificed.

MANAGEMENT OF OBSTRUCTION OF THE COLON

The management of obstruction of the colon depends upon whether the obstruction is acute or chronic complete or incomplete. The symptoms presented by the patient vary widely with the nature and degree of obstruction. Although statistically the ratio of small bowel obstruction to large bowel obstruction is approximately 4:1 the fact that the majority of colonic obstruction cases is due to carcinoma makes this type of obstruction an extremely serious one. The considerable variation in the statistical incidence of complete obstruction due to carcinoma of the colon appears to depend upon the type of surgical patient with which one comes in contact. The Lahey Clinic reports that in their experience approximately 2 per cent of all carcinomas of the colon had produced complete obstruction. At the Mayo Clinic the figure was 5.6 per cent whereas Becker reports from Charity Hospital in New Orleans found that 25 per cent of the patients admitted with carcinoma of the colon were completely obstructed.

It is generally accepted that acute obstruction of the colon due to carcinoma is a disease which requires the earliest possible intestinal decompression. Because of the nature of the obstructing



FIG. 3/6 Marked distention of the right colon in the presence of obstruction of the rectosigmoid with a competent ileocecal valve. In this type of case immediate defunctionizing colostomy is indicated.

mechanism the relief of the intracolonic pressure must be obtained by surgical means. The use of the long intestinal decompression tube with suction as the sole means of decompression in lesions of the colon particularly lesions of the left colon is absolutely contra indicated. The use of the long intestinal decompression tube does however have its place in obstructions of the ileocecal valve or in lesions of the right colon accompanied by an incompetent ileocecal valve. In such cases since the bowel proximal to the point of obstruction is the small bowel it can be completely decompressed by the use of this long tube. Such decompression in lesions beyond the cecum regardless of the status of competence of the ileocecal valve is usually not obtainable and in the case of lesions of the left colon is absolutely contra indicated.

Although Baronofsky has reported primary resection and anastomosis as a method of treatment in the presence of acute colonic obstruction it has been well established that resection of the colon in

the presence of acute obstruction is a dangerous procedure.

Decompression of the obstructed colon by incision or puncture is one of the oldest operations known to surgery. The first proposed colostomy for the relief of obstruction due to malformation of the rectum in 1710. However Pillore in 1776 was the first to perform a right inguinal cecostomy for the relief of obstruction of the rectum caused by a tumor. One of the most important factors in the management of obstruction of the colon has been the widespread use of decompression either by surgical procedure or by medical means as an aid in restoring the patient's physiologic equilibrium. When surgery is indicated decompression generally can be obtained by cecostomy for lesions of the right colon and by transverse defunctionizing colostomy for lesions of the left colon. In addition such defunctionizing and decompressing procedures may often be desirable as the first stage of a two stage operation for removal of malignant growths.

Resection of the colon usually can be accomplished by one of three different operative procedures. The choice of the operation used in any one case depends upon the condition found at the time of surgery as well as the training of the operating surgeon. The surgical methods in common use for colonic lesions follow.

- 1 Primary resection and anastomosis. This may be an end to end or an end to side anastomosis. Side to side anastomosis is rarely used. End to end anastomosis is the procedure of choice.
- 2 Obstructive resection. This operation is now used less commonly than it was in former years. With the widespread use of antibiotics and better anesthesia permitting more extensive dissection there are very few indications for obstructive resection.
- 3 The exteriorization procedure. At the present time this procedure has a limited field of usefulness.

In primary resection and anastomosis and in obstructive resection cecostomy may be employed routinely as a decompressive measure. Rankin is an ardent proponent of obstructive resection using a complementary cecostomy as a decompressive

measure. By means of preliminary cecostomy in the performance of the Rankin obstructive resection the clamp may be permitted to remain on the end of the resected bowel until it falls off. This insures against leakage and infection.

Obstruction of the Right Colon

The surgical treatment will vary considerably depending upon the nature and the condition of the bowel found at the time of surgery. In early cases with viable bowel and nonstrangulating effects if the bowel has been properly prepared with antibiotics and the bowel proximal to the ileocecal valve has been decompressed by the long intestinal decompression tube primary resection and anastomosis may be utilized. Resection of the right colon by exteriorization following the method of Mikulicz or primary resection of the right colon and ileotransverse colostomy may be performed only when the bowel or its mesentery is no longer viable.

Perforation of the Cecum Once perforation of the cecum has occurred the management of the patient becomes an extremely difficult problem. The signs of peritoneal irritation and the radiologic evidence of air under the diaphragm suggest a diagnosis of perforated viscus. In addition the association of a markedly dilated cecum is almost corroborative evidence of rupture or perforation of the cecum secondary to an obstruction distal to this point. This is considered an absolute contra-indication for exploratory laparotomy. The procedure of choice in such cases is to decompress the cecum either by means of cecostomy or if the perforations are multiple in character exteriorization of the cecum. Manipulation of a distended cecum through a large laparotomy incision in the peritoneal cavity contaminated by intestinal contents is an extremely dangerous procedure.

The safest course to follow in single perforations of the cecum has been to explore the cecum through a small transverse incision and if a perforation is found a large sized catheter should be placed in the perforation and sutured in place by purse string sutures. A second method of management in this type of case is exteriorization of the cecum if the mesentery permits no further exploration is carried out at that time.

In those cases in which no perforation of the cecum is discovered the incision can be extended transversely and exploration carried out. In this way the pitfalls and dangers of blind cecostomy will be avoided. Any patient subjected to decompressive cecostomy must be carefully observed for three or four days in order to be certain that adequate decompression of the cecum is taking place. Many deaths have resulted from perforation of the cecum which developed after surgical decompression was inadequately performed.

Carcinoma In obstruction due to carcinoma or in cases of intussusception associated with carcinoma the extent of the resection should be indicated not by the length of the intussusception but rather by the regional zone of lymphatic spread. In lesions of the right colon the right hemicolectomy with ileotransverse colostomy end to end is the procedure of choice.

Cecal Volvulus In the management of cecal volvulus once the diagnosis has been made the abdomen is opened by a right paramedian incision. The successful passage of a long intestinal decompression tube prior to surgery will do much to facilitate the operative procedure by decompressing the small bowel. However in no case should the surgical procedure be delayed in order to obtain a successful passage of the long tube. In those cases in which the intestinal tube has passed beyond the ligament of Treitz it is possible to rapidly thread the mercury weighted intestinal tube down to the ileocecal valve thus decompressing it. Once the small bowel has collapsed over the intestinal tube it may then be packed out of the way permitting a careful survey of the point of obstruction and its correction.

When the abdomen is opened in these cases the right iliac fossa should be examined and the position of cecum noted. If the cecum is not in its usual location this indicates the accuracy of the pre-operative diagnosis. The ascending colon should then be picked up and followed to the cecum. It may be found turning away toward the left almost transversely from its junction at the hepatic flexure and it may disappear under cover of the twisted mesentery. If there is marked intestinal distention the cecum may displace the coil of small bowel and tube out as soon as the

abdomen is opened. In the instances in which the distention of the cecum is moderate it may be necessary to remove many of the small bowel loops before the cecum can be seen. Once the small bowel loops have been brought out of the abdomen the small bowel mesentery undergoes detorsion by itself and the cecum then turns back on its right side. No attempt should be made to extricate the cecum from a displaced position before untwisting the mesenteric torsion.

If the cecum has become adherent detorsion may be difficult or impossible even with complete excision of small bowel. Such adhesions should be carefully separated and then the cecum carefully manipulated manually over toward the right side. A greatly distended thinned out cecum with an edematous wall is very likely to rupture. In those instances in which the cecal wall has become gangrenous rupture is almost inevitable. After untwisting the volvulus of the cecum and ascending colon the right colon must be carefully examined for signs of necrosis.

If the cecum is only moderately distended with out any necrotic areas in its wall and peristaltic activity is still present the bands fixing the hepatic flexure of the colon to the posterior abdominal wall should be cut and the ileocolic loop may be left in the abdomen after simple detorsion. This permits the right colon to fall freely from the transverse colon and removes the central pivot about which cecal volvulus occurs.

In those cases in which the cecum is tremendously distended even though necrotic patches have not yet appeared peristaltic movements may be absent. Decompression of this markedly distended cecum may be accomplished at the time of surgery by using the intestinal decompression sound. Under such conditions cecostomy using a tube about which omentum is wrapped may be desirable. In this way the cecum and right colon are fixed to the lateral abdominal wall and decompressed at the same time.

If gangrenous changes have occurred in the cecum or part of the right colon a primary resection and anastomosis of the colon with an ileo-transverse colostomy end to end is the procedure of choice. During the acute stage of the disease with a marked intestinal distention such surgery

is extremely dangerous. In those cases in which the patient will not tolerate a more extensive procedure exteriorization of the ileocolic loop may be the procedure of choice. The exteriorized loop is then amputated and a catheter inserted in the proximal ileal limb permitting intestinal decompression. When the electrolyte balance and hydration of the patient have been restored to normal the spur colostomy may be resected and an end to end anastomosis performed.

In the instances in which excision and anastomosis have not been required and detorsion has been the procedure used because distention of the cecum is not marked fixation of the cecum has been suggested. This method of preventing recurrence consists of freeing the peritoneal fold on the lateral aspect of the cecum and placing it over the untwisted portion of ascending colon suturing it to the medial aspect of the ascending colon. As a result of this the ileocecal region becomes placed retroperitoneally. This is a secure preventive measure.

Among the more uncommon anomalies associated with cecal volvulus is reversed rotation of the intestine. Approximately 18 cases of this type had been reported in the literature up to 1951. In such cases as the result of reversed rotation the transverse colon is found to emerge posterior to the mesentery of the small bowel passing through a tunnel in the mesentery of the small bowel. As a result the superior mesenteric artery and vein pass over the transverse colon. In cases of obstruction associated with reversed rotation the heavily weighted fluid containing small bowel may exert such pressure and pull upon its mesentery that it mechanically obstructs the transverse colon posterior to it even when the cecal volvulus has been untwisted and the obstruction released. The passage of an intestinal decompression tube with decompression of the small bowel results in a release of this mechanical compression of the transverse colon so that the continuity of the colon is restored.

Appendicostomy in Surgery of the Right Colon. Appendicostomy as a decompressive measure is mentioned merely to be condemned. The appendix may have been removed during previous surgery; it may be atrophic; it may be retrocecal or even when available it may have a small

measure. By means of preliminary cecostomy in the performance of the Rankin obstructive resection the clamp may be permitted to remain on the end of the resected bowel until it falls off. This insures against leakage and infection.

Obstruction of the Right Colon

The surgical treatment will vary considerably depending upon the nature and the condition of the bowel found at the time of surgery. In early cases with viable bowel and nonstringulating effects if the bowel has been properly prepared with antibiotics and the bowel proximal to the ileocecal valve has been decompressed by the long intestinal decompression tube, primary resection and anastomosis may be utilized. Resection of the right colon by exteriorization following the method of Mikulicz or primary resection of the right colon and ileotransverse colostomy may be performed only when the bowel or its mesentery is no longer viable.

Perforation of the Cecum. Once perforation of the cecum has occurred the management of the patient becomes an extremely difficult problem. The signs of peritoneal irritation and the radiologic evidence of air under the diaphragm suggest a diagnosis of perforated viscus. In addition the association of a markedly dilated cecum is almost corroborative evidence of rupture or perforation of the cecum secondary to an obstruction distal to this point. This is considered an absolute contraindication for exploratory laparotomy. The procedure of choice in such cases is to decompress the cecum either by means of cecostomy or if the perforations are multiple in character exteriorization of the cecum. Manipulation of a distended cecum through a large laparotomy incision in the peritoneal cavity contaminated by intestinal contents is an extremely dangerous procedure.

The safest course to follow in single perforations of the cecum has been to explore the cecum through a small transverse incision and if a perforation is found a large sized catheter should be placed in the perforation and sutured in place by purse string sutures. A second method of management in this type of case is exteriorization of the cecum if the mesentery permits no further exploration is carried out at that time.

In those cases in which no perforation of the cecum is discovered the incision can be extended transversely and exploration carried out. In this way the pitfalls and dangers of blind cecostomy will be avoided. Any patient subjected to decompressive cecostomy must be carefully observed for three or four days in order to be certain that adequate decompression of the cecum is taking place. Many deaths have resulted from perforation of the cecum which developed after surgical decompression was inadequately performed.

Carcinoma. In obstruction due to carcinoma or in cases of intussusception associated with carcinoma the extent of the resection should be indicated not by the length of the intussusception but rather by the regional zone of lymphatic spread. In lesions of the right colon the right hemicolectomy with ileotransverse colostomy end to end is the procedure of choice.

Cecal Volvulus. In the management of cecal volvulus once the diagnosis has been made the abdomen is opened by a right paramedian incision. The successful passage of a long intestinal decompression tube prior to surgery will do much to facilitate the operative procedure by decompressing the small bowel. However in no case should the surgical procedure be delayed in order to obtain a successful passage of the long tube. In those cases in which the intestinal tube has passed beyond the ligament of Treitz it is possible to rapidly thread the mercury weighted intestinal tube down to the ileocecal valve thus decompressing it. Once the small bowel has collapsed over the intestinal tube it may then be picked out of the way permitting a careful survey of the point of obstruction and its correction.

When the abdomen is opened in these cases the right iliac fossa should be examined and the position of cecum noted. If the cecum is not in its usual location this indicates the accuracy of the preoperative diagnosis. The ascending colon should then be picked up and followed to the cecum. It may be found turning away toward the left almost transversely from its junction at the hepatic flexure and it may disappear under cover of the twisted mesentery. If there is marked intestinal distention the cecum may displace the coils of small bowel and bulge out as soon as the

abdomen is opened. In those instances in which the distention of the cecum is moderate it may be necessary to remove many of the small bowel loops before the cecum can be seen. Once the small bowel loops have been brought out of the abdomen the small bowel mesentery undergoes detorsion by itself and the cecum then turns back on its right side. No attempt should be made to extricate the cecum from a displaced position before untwisting the mesenteric torsion.

If the cecum has become adherent detorsion may be difficult or impossible even with complete excision of small bowel. Such adhesions should be carefully separated and then the cecum carefully manipulated manually over toward the right side. A greatly distended thinned out cecum with an edematous wall is very likely to rupture. In those instances in which the cecal wall has become gangrenous rupture is almost inevitable. After untwisting the volvulus of the cecum and ascending colon the right colon must be carefully examined for signs of necrosis.

If the cecum is only moderately distended with out any necrotic areas in its wall and peristaltic activity is still present the bands fixing the hepatic flexure of the colon to the posterior abdominal wall should be cut and the ileocolic loop may be left in the abdomen after simple detorsion. This permits the right colon to fall freely from the transverse colon and removes the central pivot about which cecal volvulus occurs.

In those cases in which the cecum is tremendously distended even though necrotic patches have not yet appeared peristaltic movements may be absent. Decompression of this markedly distended cecum may be accomplished at the time of surgery by using the intestinal decompression sound. Under such conditions cecostomy using a tube about which omentum is wrapped may be desirable. In this way the cecum and right colon are fixed to the lateral abdominal wall and decompressed at the same time.

If gangrenous changes have occurred in the cecum or part of the right colon a primary resection and anastomosis of the colon with an ileo-transverse colostomy end to end is the procedure of choice. During the acute stage of the disease with a marked intestinal distention such surgery

is extremely dangerous. In those cases in which the patient will not tolerate a more extensive procedure exteriorization of the ileocolic loop may be the procedure of choice. The exteriorized loop is then amputated and a catheter inserted in the proximal ileal limb permitting intestinal decompression. When the electrolyte balance and hydration of the patient have been restored to normal the spur colostomy may be resected and an end to end anastomosis performed.

In those instances in which excision and anastomosis have not been required and detorsion has been the procedure used because distention of the cecum is not marked fixation of the cecum has been suggested. This method of preventing recurrence consists of freeing the peritoneal fold on the lateral aspect of the cecum and placing it over the untwisted portion of ascending colon suturing it to the medial aspect of the ascending colon. As a result of this the ileocecal region becomes placed retroperitoneally. This is a secure preventive measure.

Among the more uncommon anomalies associated with cecal volvulus is reversed rotation of the intestine. Approximately 18 cases of this type had been reported in the literature up to 1951. In such cases as the result of reversed rotation the transverse colon is found to emerge posterior to the mesentery of the small bowel passing through a tunnel in the mesentery of the small bowel. As a result the superior mesenteric artery and vein pass over the transverse colon. In cases of obstruction associated with reversed rotation the heavily weighted fluid-containing small bowel may exert such pressure and pull upon its mesentery that it mechanically obstructs the transverse colon posterior to it even when the cecal volvulus has been untwisted and the obstruction released. The passage of an intestinal decompression tube with decompression of the small bowel results in a release of this mechanical compression of the transverse colon so that the continuity of the colon is restored.

Appendicostomy in Surgery of the Right Colon. Appendicostomy as a decompressive measure is mentioned merely to be condemned. The appendix may have been removed during previous surgery; it may be atrophic; it may be retrocecal or even when available it may have a small

lumen which is inadequate for satisfactory decompression purposes. As a decompression measure in obstructions of the hepatic flexure appendicostomy is absolutely contraindicated. We do not believe that appendicostomy has any place in the modern management of obstruction of the colon.

Obstruction of the Left Colon

The surgical management of obstruction of the left colon due to adhesive bands may be simply corrected by lysis of such bands. Obstruction of this type is uncommon in the left colon but has been reported occasionally as a result of previous surgery or as a result of organization of intra-abdominal hemorrhage. The surgical management of obstruction of the left colon depends to a great extent upon the degree of obstruction present as well as the type of obstructing mechanism. In order of frequency the most common causes of



FIG 377 The use of the intestinal decompression tube preliminary to resection of the left colon for carcinoma. This is useful in those cases associated with an incompetent ileocecal valve.



FIG 378 Same patient as in Figure 377. Note obstruction of the descending colon. Successful decompression of the small bowel renders resection and anastomosis of the obstructed colon a much simpler procedure.

obstruction of the left colon are carcinoma, diverticulitis, and volvulus—the last two ranking a poor second and third.

Carcinoma. In recent years primary resection and anastomosis without colostomy has been advised in obstructions of the left colon due to carcinoma. Although many cases have been treated successfully in this fashion, the method is still a dangerous one. The performance of a preliminary defunctionizing transverse colostomy is recognized as a first stage procedure in the management of this disease. Following defunctionizing colostomy with the colon adequately prepared and cleaned, a resection of the malignant growth producing the obstruction plus the entire gland-bearing area should be the treatment instituted. This may require ligation of the inferior mesenteric artery at the aorta and resection of the entire left colon with anastomosis of the transverse colon to the upper rectum. Unless an adequate resection is

performed the patient is doomed from the very beginning. In those cases in which it is thought that the transverse colon may have to be anastomosed to the rectosigmoid, a colostomy may be used as a decompressive measure. Although true defunctionizing of the colon is not obtained by this method, the placement of the colostomy at this point permits the free use of the transverse colon in the anastomosis.

In obstruction of the left colon in which liver metastasis has already occurred and in which a cure is out of the question, a limited palliative resection may be performed in order to re-establish the continuity of the bowel.

Diverticulitis. The widely accepted method of treating diverticulitis of the left colon causing obstruction is a three-stage surgical procedure.

First Stage. In the first stage the transverse defunctionizing loop colostomy is performed preferably transecting the medial half of the left rectus muscle. One should attempt to stay away from the right rectus muscle so that this area may be left clear in the event the patient requires biliary or gastric surgery at some later date. One objection to transecting the left rectus in the performance of colostomy is the fact that if the diagnosis is in error and the obstruction is due to carcinoma, that portion of the transverse colon may be lost to the body as a point which may be anastomosed to the rectosigmoid. For this reason there are many surgeons who transect the right rectus in all cases and bring the colostomy through that area. In any case in which the diagnosis is in doubt this would be the preferable procedure. In addition, in those cases in which the splenic flexure is very highly placed or where angulation is apt to occur the surgeon has no choice but to utilize the right half of the transverse colon. In all cases the colostomy loop must be completely cut across. Only in this way can an almost complete diversion of the fecal stream be obtained. With the proper use of antibiotics, rest and defunctionizing colostomy the inflammatory process usually subsides. This is particularly true if the inflammatory obstructing mass of diverticula presents a large inflammatory process or has perforated with abscess formation. Under these circumstances a complete diversion of the fecal stream is imperative.

Second Stage. The second stage in the procedure is resection of the obstructed bowel. The length of time permitted to elapse between the performance of the colostomy and the time of resection and anastomosis is variable, depending upon the extent of the inflammatory process and the condition of the patient. Generally at least six months should be permitted to elapse although in those cases in which the diagnosis is in error and a carcinoma is present such a lapse would not be wise. It is for this reason that during the performance of the colostomy a careful examination or survey of the point of obstruction should be made as well as a possible biopsy if the lesion is accessible. The inflammatory process should be permitted to resolve completely before primary resection and anastomosis is performed. The only exceptions to this rule are those instances in which there is still a discharge of bloody mucus per rectum despite the fact that complete diversion of the fecal stream has occurred after the first week. This is highly suggestive of the presence of a carcinoma of the colon either with or without diverticulitis. Usually following a defunctionizing colostomy for diverticulitis there is little or no discharge per rectum after the first week.

In performing an anastomosis and resection for diverticulitis the anastomosis must be made between bowel ends that are free of inflammation. This may mean that the distal transverse colon may be anastomosed to the rectosigmoid. Under no circumstances should the anastomosis be made between inflamed or scarred colon. It is not unusual to find that the entire left colon may be scarred and narrowed as a result of the diverticulitis. Under such circumstances the whole area should be eliminated.

In the event that an associated carcinoma is suspected a radical surgical approach to this area must be performed as soon as the patient has recovered from the obstructive symptoms. This may be as early as the second or third week after colostomy. In those cases in which it has been established that the obstructive lesion is purely inflammatory in nature a period of six months or longer may be permitted to elapse before resection and anastomosis.

Third Stage. Following the primary resection

PREVENTION OF ADHESIONS

It is very difficult to prevent the formation of postoperative adhesions between damaged visceral surfaces. The tendency to form such adhesions varies with the individual. Often adhesions of such extensiveness develop following abdominal surgery that the patient is subjected to repeated attacks of intestinal obstruction throughout the rest of his life. Numerous operative procedures performed to relieve the obstruction may only aggravate the situation by producing increased involvement. The individual may become an abdominal cripple suffering almost continually from pain and varying degrees of bowel obstruction. Furthermore, since eating irritates increased peristaltic activity and hence abdominal discomfort, there may be a great weight loss due to anorexia.

It has been repeatedly demonstrated that simple angulation of a loop of bowel in itself does not result in obstruction. Obstruction is likely to occur at the site where a loop of bowel has become adherent to the pelvic wall or to the root of the mesentery and becomes sharply angulated at its point of attachment. Edema at these points of attachment when coupled with the sharp angulation of the bowel readily creates an acute obstruction in an already narrowed bowel lumen.

There has been considerable experimental work performed in an effort to prevent the formation of these intraperitoneal adhesions. This work has proceeded along four lines:

- 1 Avoidance of technical errors which may cause adhesions
- 2 Intraperitoneal instillation of substances to prevent adhesion formation

- 3 Injections of hormones or drugs to limit adhesion formation

- 4 Operative treatment to prevent recurrent obstruction by controlling the site of formation of the adhesions

AVOIDANCE OF TECHNICAL ERRORS WHICH CAUSE ADHESIONS

It has been proved that the talc used in preparing rubber gloves is a peritoneal irritant and is the cause of many of the postoperative adhesions. Consequently, nonirritating starch powders have been substituted for talc in the preparation of these gloves. The gloves are also rinsed in sterile water prior to the introduction of the surgeon's hand into the peritoneal cavity.

Vaughn has summarized the important points in the prevention of adhesions as follows:

- 1 Careful handling of the tissue, particularly the bowel and peritoneum in order to prevent any loss of continuity of the peritoneal covering
- 2 Eversion of the parietal peritoneum of the incision with small caliber plain catgut
- 3 Complete hemostasis so that no blood clotting is or will be present as a possible precursor of adhesions
- 4 If possible, peritonealization of all surfaces on the intestine and the anterior abdominal wall
- 5 Avoiding the use of talc powder on the gloves
- 6 Washing the gloved hand frequently in sterile water to remove any blood or debris
- 7 Careful and accurate approximation of the tissues in gastric and intestinal surgery to

- prevent any leakage which would invite an acute or subacute inflammatory process with a subsequent formation of adhesions
- 8 Warm moist saline laparotomy sponges when examining or packing away the bowel
 - 9 Antibiotics postoperatively to suppress the infection arising from contamination
 - 10 Early ambulation when possible or changing the position of the patient frequently while in bed
 - 11 Early feeding to stimulate peristaltic activity
 - 12 Cutting all tied sutures short avoiding long tails to which a loop of bowel might become adherent

A delay in the recognition and institution of proper surgical treatment for intestinal obstruction due to adhesions may set the stage for such extensive inflammatory intra abdominal disease that chronic intestinal obstruction may result. At the first operation for obstruction the adhesive band or bands should be cut. If this is done early in the course of the disease the patient recovers promptly and usually no further obstruction occurs. Additional factors which will aid in the reduction of postoperative adhesions include the avoidance of tissue trauma and the elimination of such foreign bodies as drains and chemical irritants. The installation of a penicillin solution may be of some value by reducing infection.

INTRAPERITONEAL INSTILLATION OF SUBSTANCES TO PREVENT ADHESIONS

Among the substances used to prevent intraperitoneal adhesions are amniotic fluid, saline, air, oil, blood, gum, acacia, proteolytic enzymes, papain, trypsin, and heparin.

In 1942 Boys critically evaluated the methods recommended for the prevention of peritoneal adhesions. All of the methods previously proposed were found to be unsatisfactory with the possible exception of the intraperitoneal instillation of heparin. Although this method seemed to be effective in experimental animals it had a limited value in humans since there appeared to be a definite danger of massive intraperitoneal hemorrhage following its use. Bloor and his associates studied this problem extensively in an attempt to evaluate

the effect of heparin in controlling the formation as well as the reformation of adhesions in rabbits. From their studies they concluded that heparin was not effective from either point of view. Furthermore many of the rabbits succumbed as a result of intraperitoneal hemorrhage and hemorrhages into vital organs. As a result of these studies it was concluded that although the use of heparin in the prevention of adhesions was based upon its prevention of blood coagulation in the peritoneal cavity, in actual practice heparin did not retard the formation of peritoneal adhesions and was undesirable because of the danger of intraperitoneal hemorrhage.

Experiments similar to those performed with heparin were also performed with Dicumarol. This anticoagulant also failed to prevent intraperitoneal adhesions. These observations suggest that blood coagulation alone is not necessarily essential to intraperitoneal adhesion formation.

Papain has been used with success by some surgeons. This is a proteolytic enzyme which does not attack viable tissue. It was used to prevent the formation of adhesions due to postoperative exudation by causing digestion of such exudation. Practically the distribution of papain in the peritoneal cavity was found to be inconstant and its mixture with the blood and serum which are present in the peritoneal cavity limits its effectiveness.

At present it appears to be generally agreed that none of these substances used intraperitoneally to prevent adhesions are effective clinically.

INJECTIONS OF HORMONES OR DRUGS TO LIMIT ADHESION FORMATION

Among the methods proposed to prevent postoperative adhesions, daily intramuscular injections of cortisone acetate for 7 days before and 16 days after the experimental production of talc induced adhesions have been shown to be of value. From this Lyall concluded that in guinea pigs adhesion formation might be prevented by the parenteral use of cortisone. It was not certain, however, whether this could be profitably applied to humans because of the possible harmful effects of cortisone on the healing of the abdominal wound. There is still some doubt about the exact method by means of which cortisone prevents adhesion formation. The obvious explanation would be the power of

retarding fibroplasia but there is some evidence of other possible factors. The amount of clear peritoneal exudate which appears to be increased by such injections may contain some spreading factor which prevents adhesion formation. In addition the increased motility of the gastrointestinal tract may play a part.

Shenbrot and Saltzman studied the effects of cortisone and corticotropin (ACTH) on intra-abdominal adhesions in experimental animals. They found that the cortisone and the corticotropin were effective in reducing the number of intraperitoneal adhesions which resulted from talc produced adhesions in dogs and rats. Dosages of 10 mg of cortisone or 5 mg of corticotropin twice a day in the dog and 2 mg of cortisone once a day in the rat were sufficient to prevent the formation of adhesions. The dose was not large enough to interfere with wound healing and there was not a single incident of wound dehiscence or abdominal evisceration. These authors believed that daily injections of cortisone and corticotropin for 10 to 14 days were necessary for the prevention of intra-abdominal adhesions in experimental animals.

Ragan held that the suppression of healing in his series of cases during cortisone and corticotropin therapy was a general phenomenon resulting from the action of these hormones on all mesenchymal tissues and should lessen the formation of adhesions in any part of the body. Cortisone had previously been used by Stuckfield to prevent joint adhesions following arthroplasty. Whether the use of cortisone or corticotropin would be practical in the prevention of intra-abdominal adhesions in humans depends upon the difference in dosage between that required to prevent adhesions and that which would delay wound healing. It seems that the undesirable effects of cortisone and corticotropin were more directly related to the total amount of drug given than to the size of the daily dosage. An additional factor seems to be the fact that the use of cortisone and corticotropin might mask pain, diminish muscular rigidity and suppress fever. Because of this postoperative complications might easily be overlooked. In addition it would appear that the use of cortisone or corticotropin would be dangerous in those cases in which any intestinal surgery had been

done because of the danger of a blowout at the point of anastomosis due to impairment of the fibroblastic proliferative process required for healing. Furthermore prolonged use of cortisone or corticotropin has on occasion been known to produce perforation of pre-existing gastric or duodenal ulcers as well as perforation of ulcerative colitis. From these observations it would appear that the use of cortisone or corticotropin might be dangerous in those cases in which there has been bowel damage where fibroblastic proliferation is required for repair.

Prostigmin has been used to prevent postoperative adhesions. Schiff and his associates found in some experimental studies that when postoperative adhesions were dissected free, they were far less likely to grow back if motility of the bowel was maintained by early feeding and injections of Prostigmin. This regime is particularly suitable for the postoperative management of those patients in whom extensive dissection was required. It is not suitable for use in those cases in which bowel resection had been performed because of the danger of leakage at the line of anastomosis caused by the return of vigorous peristalsis before adequate healing had occurred. In an experimental study on dogs Schiff found that only 33 per cent of the cases treated by early feeding and Prostigmin formed adhesions following talc applications to the peritoneal cavity as compared with 92 per cent in a series of animals in which no Prostigmin or early feeding was employed. In this latter group of animals atropine sulfate was given orally as an additional means of inhibiting gastrointestinal motility. These experimental findings suggest that early feeding and Prostigmin or other means of increasing intestinal motility postoperatively provide a rather effective method for the prevention of postoperative adhesions. This method is especially useful in the management of those cases in which surgery has already been performed for intestinal obstruction due to adhesions and in which the adhesions had been cut.

SURGICAL METHOD OF PREVENTING OR SELECTIVELY FORMING ADHESIONS

Thomas and Rhoads attempted to prevent the formation of adhesions in overlying segments

of bowel in which the serosa had been removed. In their experimental study they deliberately denuded serosal areas from the bowel of rats and guinea pigs. By oversewing this denuded area they hoped to lower the incidence of adhesion formation. However these attempts to prevent adhesion formation failed and it was found that the oversewing procedure had little or no effect on the prevention of adhesions.

Noble Plication Procedure

Among the practical methods of controlling the formation of adhesions is the operation of plication of the small bowel first described by Noble in 1937. This operation developed from the concept that while the formation of peritoneal adhesions following surgery, trauma or inflammatory intra abdominal processes could not be prevented the site of their formation could be controlled in such a fashion that the eventual scar tissue would not constrict the lumen of the bowel thus obstructing it. To this end Noble devised an operative procedure which purposely created adhesions in an orderly fashion and in such a manner that obstructions would not be likely. Subsequently other surgeons have suggested the use of the Noble plication procedure in cases of recurrent intestinal obstruction due to adhesions. The literature indicates that the use of this procedure has been quite satisfactory, especially in the management of those cases which have been subjected to multiple operations for lysis of adhesions. Although occasional bad results have been reported there seems to be general agreement that in those cases which have been previously subjected to multiple surgery for the lysis of adhesions this procedure may be a preventive method since with this technique adhesions are formed at a site elected by the surgeon rather than at random. Attention and painstaking adherence to some of the technical details of the operation may greatly decrease the poor results associated with the Noble plication procedure.

The indications for the Noble plication procedure follow:

- 1 The presence of massive adhesions in the peritoneal cavity which obstruct the lumen of the small bowel. These adhesions may

have been produced by previous surgery for such acute inflammatory abdominal processes as pelvic infections, appendicitis or perforated peptic ulcer.

- 2 The symptoms of chronic intestinal obstruction such as cramping abdominal pain, weight loss and on occasion diarrhea.
- 3 Chronic intestinal obstruction with its associated abdominal discomfort to prevent morphone addiction.
- 4 To prevent the reformation of adhesions which may obstruct the lumen of the bowel.
- 5 Those cases presenting a matting syndrome in which loops of bowel are extensively matted to each other and to the anterior abdominal wall. In such cases a tedious dissection of the intestinal loops is required followed by a Noble plication to form selective adhesions without obstruction.
- 6 Those patients in whom intestinal obstruction has not occurred but where in the course of the surgical procedure for some other intra abdominal pathology the small bowel is found to be so matted together by multiple adhesions that separation of these adhesions is required leaving many raw serosal surfaces. In these cases although intestinal obstruction had not been present before it is quite likely that this complication may occur at any time in the future. To avoid this possibility the bowel may be plicated in the pattern suggested by Noble.
- 7 Those cases of acute intestinal obstruction in which during the course of the dissection large areas of serosa are denuded. Since these denuded surfaces which are the potential source of adhesions are too extensive to be resected primarily without the risk of causing severe nutritional deficiencies they are brought together via the plication procedure in such a fashion that angulation and acute mechanical obstruction cannot occur.

The technique of surgery in the performance of a proper Noble plication procedure is as follows:

- 1 A careful and painstaking dissection of all adhesions using sharp dissections with a scalpel in order to avoid unnecessary trauma

to the serosa and possible perforation of the bowel. During the performance of this type of dissection an adequate blood replacement must be given because there is generally considerable blood loss.

- 2 After the loops of bowel are freed from themselves and from the anterior abdominal wall, they are sutured together with a continuous absorbable suture on the mesenteric border in three or four large loops leaving the ends free to prevent sharp angulation.
- 3 The suture should begin and end 3 cm from each end in order to keep the angles gentle.
- 4 The loops should be made long enough to fit freely across the peritoneal cavity.
- 5 The bowel should be securely sutured with 000 chromic catgut. A continuous stitch should be used to unite the opposing loops of bowel to each other.
- 6 If the entire small bowel is not plicated the folds of the mesentery must be closed in order to prevent loops of bowel from entering the sac formed by the mesentery.
- 7 Adequate plication must be made to cover all the raw surface areas.
- 8 The bowel should be stimulated to peristaltic activity following surgery. Noble believed that vigorous postoperative bowel stimulation should be carried on until liquid stools occurred.

The use of nonabsorbable interrupted sutures may lead to perforation of the bowel. Cases of this accident have been reported. To be successful the plication must include all of the intestine which is involved; this can sometimes include all of the small bowel from the ileocecal valve to the ligament of Treitz. Radiologic follow up studies in these cases demonstrated that the plicated intestines functioned normally.

Although it is agreed that the Noble plication procedure is the logical method for controlling the formation of intra abdominal adhesions in those cases repeatedly subjected to surgery for adhesive obstructions, great care must be taken in the selection of cases to undergo this operation. It might be desirable to subject all cases to neuropsychiatric study prior to the institution of the Noble corrective measure in order to rule out the neuropsychi-

atric patient, the psychoneurotic, and the dope addict.

Although the recent literature recommending the use of the Noble plication procedure reports generally favorable results, failures have been reported in approximately 10 per cent of the cases so treated. In some cases this is due to an error in technique. In one case reported by Seabrook and Wilson, in a second operation three months after the first, the entire mass of plicated bowel was found to have undergone volvulus producing acute intestinal obstruction. At operation the entire twisted mass was delivered from the pelvis and covered with omentum after being untwisted. The patient made an uneventful recovery. In an occasional case the plicated loops may separate causing obstruction. Another possible complication to the Noble plication procedure is a small bowel fistula as a result of slough of the suture holding the loops together. This is particularly apt to occur in those cases in which interrupted sutures of the nonabsorbable type are used. In the series of Noble plication procedures reported by Seabrook and Wilson, there were four postoperative small bowel fistulas. In the series of cases of Noble plication reported by Boys, 8 per cent developed a small bowel fistula. In two of the patients who developed a fistula, death occurred as a result.

In addition to the mortality which may be due to technical errors, the placing of sutures or the formation of fistulas, the procedure itself may contribute to the mortality rate because of the extent of both primary disease and the surgical treatment. Although Noble suggested plicating the bowel at its mesenteric border, Seabrook and Wilson suggested that the suture should be placed midway between the mesentery and the antimesenteric border. They believe that if the antimesenteric edges of the bowel were sutured to, either to form the loops, violent string adhesions would occur as a result of the small bowel becoming distended. Such distention would cause the adherent loops to pull apart. On the other hand, if the loops of bowel are sutured at the mesenteric border, the loop may fall over and attach to the mesentery causing an acute angulation. An additional complication of the extensive dissection required in a Noble plication procedure is the attachment of the

small bowel to the abdominal incision thus producing kinking and obstruction. In conclusion it should be added that any patient on whom a Noble plication procedure was performed who later develops abdominal pain or signs of obstruction should be operated upon without delay because the mortality rate is very high when this is not done.

Satinsky Kron Method of Plication

Satinsky and Kron proposed an alternative method to the Noble plication procedure in the management of those cases subjected to multiple surgery for recurrent intestinal obstruction. Their main objection to the Noble method was that they did not believe it assured an adequate lumen in the immediate postoperative period. In addition they called attention to the fact that obstruction may occur from edema constricting adhesions kinking or inflammatory changes. Leakage at the points of suture with resultant peritonitis and fistula which sometimes result from the Noble plication procedure are also mentioned by these authors as further disadvantages to this method.

Satinsky and Kron propose what they believe to be a better method which involves the mobilization of the entire small bowel relieving all mechanical causes of obstruction. A long piece of semi rigid rubber tubing 6 mm. in luminal diameter and containing spirally placed perforations every $1\frac{1}{2}$ inches is inserted by way of the nostril and threaded through the small bowel into the cecum. The proximal end of the tube is secured to the nose by suture. The distal end is brought out through a cecostomy or an ileostomy or is fixed in the cecum by a balloon device.

The entire small bowel is thus pleated along the tube in an accordion like fashion. The tube is held

in position for several days until adhesion formation or peritoneal reaction fixes the bowel. This assures an adequate lumen. By applying constant suction the multiple perforations decompress any areas preventing a closed loop obstruction. The tube is permitted to retract after about a week but is not removed for three or four weeks.

In those cases in which the tube cannot be maneuvered through the duodenum a Witzel jejunostomy is used to introduce the tube into the small bowel. If an ileostomy has been performed closure of the ileostomy should not be performed until normal bowel function is insured. The authors proposed this method as a useful procedure when decompression by the Miller Abbott tube is unsuccessful.

Tube decompression is not designed to replace operation if the primary lesion causing recurrent obstruction can be removed, but rather Satinsky and Kron proposed this method to be used when the causative factors cannot be adequately dealt with. They believed the procedure would be successful when no other form of therapy was feasible for the relief and prevention of recurrent obstruction due to adhesions. They believed this method to be of particular use when the entire small bowel is edematous red and friable with complete matting, volvulus, internal herniation or kinking.

During the many years in which we have been interested in the management of intestinal obstruction cases of all kinds have gone through the surgical department at Grace Hospital. However in this period of time we have never had recourse to the procedure proposed by Satinsky and Kron. Furthermore we do not believe that this method is useful except in extremely exceptional circumstances.

ANESTHESIA IN INTESTINAL OBSTRUCTION

The importance of good anesthesia in the surgical treatment of a patient with intestinal obstruction cannot be underestimated. However the type of anesthetic given to any patient is of less importance than the ability of the anesthetist who gives it. A well trained medical anesthetist may be allowed a wide latitude in the selection of the anesthetic agent to be employed. Since he is a specialist in his own right he can select the anesthetic which is most desirable for a specific case. Unfortunately the number of anesthesiologists throughout the country is still relatively small so that less qualified personnel are often responsible for the selection and administration of the anesthetic. For this reason the surgeon should be acquainted with the various types of anesthetic which may be used in any case of intestinal obstruction.

The specific anesthetic to be used in any case of intestinal obstruction depends upon many factors. Although there are certain cases in which a specific anesthetic is definitely indicated in general the range of choice of the anesthetic for any one case is wide and depends to a considerable extent upon the individual preference of the anesthesiologist. The anesthetic agents used in the management of intestinal obstruction are local anesthesia, spinal anesthesia and general anesthesia.

LOCAL ANESTHESIA

This form of anesthesia is useful in the management of the poor risk patient whose condition is so bad that only a minimal procedure such as enterostomy or colostomy is to be performed. This type of patient may be admitted to the hospital with marked intestinal distention, poor electrolyte balance and in such poor condition generally that

definitive surgery is contra indicated. In these cases a properly performed local anesthetic skillfully administered permits the surgeon to perform a colostomy or enterostomy which may be a life saving procedure. Intercostal block is an excellent form of anesthesia in the hands of a skillful anesthesiologist. At times the surgeon does all the local infiltration or the anesthesiologist may perform the preliminary intercostal block as well as anesthetizing the abdominal wall while the surgeon injects the deeper structures as the surgical procedure proceeds. Adequate local anesthesia supplemented by intercostal block gives the best possible results even in the poorest risk patient. However, a local anesthetic supplemented by small intravenous injections of morphine is an extremely dangerous procedure in any patient with intestinal obstruction and distention. Morphine or Demerol produces a depression of the already depressed respiratory center which may result in serious consequences to the patient. In addition when one undertakes an abdominal procedure under a local anesthetic adequate blocking should be performed at the onset. Injecting a local anesthetic with the idea of doing as much as possible under this and then administering a general anesthetic if one is unable to continue is an extremely poor form of treatment. Under such conditions faced with the problem of anesthetizing an apprehensive patient in considerable pain the anesthesiologist must administer a larger amount of anesthetic agent for the induction of anesthesia. In the poorest risk patient this alone may be sufficient to produce fatal consequences. It must be emphasized that only minimal surgical procedures may be performed under a local anesthetic.

A local anesthetic should be used in the performance of a cecostomy without an exploratory. It carries only a minimal risk and the operative procedure may be performed both rapidly and effectively. Similarly when the condition of the patient will not permit a general anesthetic a local may readily be used in the performance of a de-functionizing loop colostomy. Local anesthesia is also indicated for the surgical treatment of strangulated hernia in the poor risk patient. The limited exposure usually obtained with local anesthesia increases the operative hazard in long standing obstructions with markedly distended loops and makes careful abdominal surgery impossible.

The use of local anesthesia is therefore of limited usefulness in the management of intestinal obstruction although it does have its place

SPINAL ANESTHESIA

Many anesthesiologists advocate the use of spinal anesthesia in cases of intestinal obstruction. In general spinal anesthesia is useful in the surgical management of intestinal obstruction and for a long time it was considered to be the anesthetic of choice. However there are many anesthesiologists who believe that it is contra indicated in any patient with an incompletely decompressed intestinal tract.

A major advantage of spinal anesthesia in the management of intestinal obstruction is the greater relaxation which it produces. This facilitates the surgical procedure by providing better exposure. Because it paralyzes the inhibitory or splanchnic nerves spinal anesthesia permits a preponderance of vagal impulses to reach the small bowel increasing peristaltic activity. Because of this effect upon intestinal motility spinal anesthesia has been recommended as a therapeutic measure in the treatment of paralytic ileus. However the short duration of this effect makes such a measure only temporarily effective. At times the increased motility resulting from spinal anesthesia is a mixed blessing. Isolated cases have been reported in which rupture of an already damaged bowel has occurred because of increased peristaltic activity.

The disadvantages to the use of spinal anesthesia

in the management of intestinal obstruction are as follows:

- 1 To be effective in patients with marked distention the spinal anesthesia must reach an area sometimes up as far as the third or fourth thoracic spinal segment. Under such conditions all the muscles used in breathing, except the diaphragm, become paralyzed and hence useless. Even though oxygen is given during the course of the operation these patients may have considerable difficulty in breathing or in sufficiently oxygenating. In addition to the loss of function of the muscles used in breathing the diaphragm is limited in its excursion by the distended loops of small bowel. The high diaphragmatic level not only decreases the breathing capacity of the patient but it also tends to produce a basal atelectasis and the diminished excursions of the diaphragm reduce the tidal volume. This combination of effects tends to produce an inadequate exchange of gases in the lungs. Consequently there may be an accumulation of excessive amounts of carbon dioxide in the blood. As a result there is a possibility that in the course of the intestinal distention the tidal exchange may be inadequate to provide sufficient oxygenation. When this happens a state of hypoxia with carbon dioxide retention tends to occur which then proceeds to asphyxia.
- 2 Spinal anesthesia if administered to an already hypotensive patient may further reduce the blood pressure. The consequences may be serious. The conditions under which this may occur are as follows:
 - a Patients brought to the operating room with long standing intestinal obstruction associated with marked intestinal distention.
 - b Patients who are cyanotic or those with labored respirations.
 - c Patients found to be dehydrated and in poor electrolyte balance due to vomiting, the indiscriminate use of gastro intestinal suction or the promiscuous drinking of

- water during the time that suction is in progress
- d Patients with high intestinal obstruction may come to surgery with alkalosis whereas patients with low intestinal obstruction may present a picture of acidosis due to starvation
 - e Many patients come to surgery in mild or moderate shock. The use of spinal anesthesia tends to increase the degree of shock

In conclusion one might say that patients in relatively good condition may be advantageously operated upon under a spinal anesthetic but that this type of anesthesia should not be used in the markedly distended patient whose condition is questionable

GENERAL ANESTHESIA

In this group we find two types of anesthesia—*inhalation anesthesia* in which one of the anesthetic gases are used, and *intravenous anesthesia*

Intravenous Anesthesia

Intravenous anesthesia using Pentothal or Nembutal either alone or in combination with curare has very little place in surgery for intestinal obstruction. These drugs are respiratory and circulatory depressants and do not produce the relaxation which is required for intra abdominal surgery of this type. Although these drugs may be combined with curare to produce the desired degree of relaxation this combination is very dangerous when given to patients in shock or in poor condition. Among the serious consequences of this type of anesthesia are the occasional reports in which as a result of the curare and Pentothal anesthesia the superior esophageal sphincter relaxes so much that a flood of gastric contents rushes forth passing up the esophagus and down into the trachea literally drowning the patient before an endotracheal tube can be inserted

Inhalation Anesthesia

Inhalation anesthesia particularly when used with an endotracheal tube is the anesthetic agent of choice. The inflation of the balloon of the endotracheal tube prevents aspiration of gastric con-

tents into the trachea or bronchial tree. Such aspiration by patients going under anesthesia is common. In a series of patients undergoing elective surgery who had nothing by mouth the evening before surgery a survey demonstrated that in the horizontal position 24 per cent regurgitated and of these 16 per cent aspirated gastric contents sometime during the course of the general anesthesia. In the poor risk patient the endotracheal tube may be introduced under local anesthesia spraying the cords and pharynx with Pontocaine or cocaine, without the preliminary use of Pentothal. With this tube in place gas oxygen ether cyclopropane or ethylene are well tolerated by the patient. In the cardiac patient the use of oxygen ether is especially desirable.

The comparative action of the various anesthetic gases upon the propulsive motility of the small bowel has been extensively studied. Nitrous oxide cyclopropane ethylene ether and chloroform were studied. A marked variability in the results was noted by different workers. In general however, chloroform and ether produced the most marked depression in intestinal motility. Bisgard and Johnson noted a correlation between the degree of gastro intestinal motor inhibition during anesthesia and the frequency and severity of post operative symptoms in surgical patients. From their experiments they were able to conclude that there was a close correlation between the degree with which gastric and intestinal motor activity is inhibited during anesthesia and the frequency duration and severity of postoperative symptoms of gastro intestinal dysfunction. Anoxia has been suggested as a factor in the inhibition of the intestine by the anesthetic gases. Apparently this is not the mechanism in cyclopropane anesthesia. Both stimulating as well as depressant effects have been ascribed to this gas by different workers. Burstein suggested in his studies that intestinal activity is inhibited in the lower planes of cyclopropane anesthesia.

The experimental studies of Plant and Miller have indicated that the action of the anesthetic gases may be peripheral. This action appears to be dependent upon extramural nervous control. Ether chloroform and deep ethylene anesthesia were depressant to Thury Vella loops created in

experimental animals. Light ethylene and nitrous oxide-oxygen anesthesia produced a marked initial increase in activity which may have been due to partial asphyxia as suggested by other workers.

The difficulty in properly evaluating the effect the anesthetic gas has upon intestinal motility is complicated by the fact that following any laparotomy intestinal motility is abolished for at least four hours regardless of anesthesia. If the intestines are cut activity may be abolished for 48 hours.

CARE OF PATIENT BEFORE ANESTHESIA

Before any patient is taken to surgery and given an anesthetic it is essential that a Levin tube or a long intestinal decompression tube be placed in the stomach and adequate suction applied to not only empty the stomach prior to surgery but to keep it empty during the course of surgery. Many patients brought to surgery with an empty stomach may regurgitate large amounts of intestinal contents into the stomach and then up through the esophagus and into the trachea. Serious pulmonary complications may be caused in this way. A review of patients in whom a properly functioning Levin tube was placed prior to the induction of anesthesia revealed that the incidence of vomiting or regurgitation was reduced 30 per cent and the incidence of aspiration of gastric contents into the lungs was reduced 66 per cent.

The presence of a long intestinal decompression tube far down the gastro intestinal tract is no assurance against the aspiration of intestinal contents in the fashion outlined. However, those patients in whom an endotracheal tube fitted with a balloon has been inserted as a method of anesthesia are singularly immune to aspiration of intestinal contents. In those cases in which a long intestinal decompression tube is far down the gastro intestinal tract it may be necessary to pass a Levin tube through the other nostril and empty the stomach just before the induction of anesthesia. The Levin tube may then be removed after com-

pletion of the surgery. In all cases of intestinal obstruction associated with much distention of the gastro intestinal tract a properly functioning Levin tube and an empty upper gastro intestinal tract are essential in any patient brought to surgery for a general anesthetic.

CARE OF PATIENT AFTER ANESTHESIA

Following surgery prior to the removal of the endotracheal tube the nasopharynx and pharynx should be thoroughly suctioned out. Following the removal of the endotracheal tube in those patients in poor condition it is highly desirable to pass a bronchoscope so that any secretions remaining in the tracheobronchial tree can be removed by suction under direct vision at that time. It is amazing how often large mucous plugs are found in the trachea which can be readily removed through a bronchoscope. Following adequate cleansing of the tracheobronchial tree the patient should be moved from the operative table to the cart with as little trauma as possible. Bouncing such patients from the operating table to the cart or from the cart to the bed may have serious consequences. Many patients in poor condition have been thrown into shock by too rough handling in their transfer.

It is dangerous to send a patient with marked abdominal distention back to his room without providing constant supervision and a suction machine at his side. In hospitals equipped with a recovery room these measures are provided. Even though the cause of the intestinal obstruction has been released by lysis of an adhesive band or correction of the obstructing mechanism during recovery from anesthesia the patient may vomit and aspirate large amounts of intestinal contents before suction equipment can be used to aspirate the pharynx. Under such conditions the stage is set for aspiration pneumonia. Many old or debilitated patients have been lost in this way even though the surgical procedure itself was successfully performed.

INTESTINAL OBSTRUCTION AFTER GASTRIC AND COLONIC SURGERY

There are many types of bowel obstruction which can result from gastric or colonic surgery. Such obstructions may be classified into two large groups: (1) the nonspecific type of small bowel obstruction and (2) the specific type of small bowel obstruction. The nonspecific type of obstruction refers to all varieties of small bowel obstruction not specifically due to gastric or colonic surgery *per se*. Patients in this group can develop small bowel obstruction simply by virtue of the fact that a laparotomy has been performed. The specific kind of surgery performed is not a factor.

The specific type of small bowel obstruction refers to all varieties of small bowel obstruction which occur as a result of the anatomic changes that have been brought about by the gastric or colonic surgery *per se*. These patients develop small bowel obstruction because of the specific surgical procedure performed.

NONSPECIFIC SMALL BOWEL OBSTRUCTION AFTER GASTRIC AND COLONIC SURGERY

Any patient subjected to laparotomy is a potential candidate for this type of obstruction. These obstructions may follow any abdominal procedure and are not characteristic of gastric or colonic surgery. All patients in this group fall roughly into two classes based upon the mechanism of obstruction. The first is paralytic ileus and the second mechanical obstruction.

Paralytic Ileus

All patients subjected to abdominal surgery are apt to develop a paralytic ileus. This varies in

degree from the mild postoperative variety of two to three days' duration to the most extreme type associated with peritoneal infection. The physical and radiologic findings are those common to paralytic ileus from any cause. The silent distended abdomen is quite characteristic. The correlation of this clinical finding with the radiologic demonstration of a small bowel and colon filled with gas and fluid is almost pathognomonic. Early in the postoperative period, however, errors in diagnosis may occur because of an associated mechanical obstruction masked by the atony of the small bowel.

In addition to the paralytic ileus of mild degree which is the physiologic reaction of peritoneal trauma, infection within the peritoneal cavity causes a persistence and accentuation of this ileus. For a complete discussion of peritoneal infection as a cause of paralytic ileus, the reader is referred to Chapter 8.

Mechanical Small Bowel Obstruction

The majority of small bowel obstructions following abdominal surgery occur as a result of postoperative adhesions. The mechanism by means of which such adhesions cause small bowel obstruction varies greatly. The following varieties have been reported:

- 1 A loop of bowel may become adherent to the incision in the abdominal wall and become obstructed.
- 2 A loop of bowel may become adherent to the abdominal wall and undergo volvulus.
- 3 A loop of small bowel may slip through or around a loop of bowel attached to the ab-

- downward wall and cause small bowel volvulus
- 4 Adhesions may so compress a loop of bowel against the bony pelvis or solid organs of the body as to obstruct it
- 5 A loop of bowel may be kinked by adhesions and become obstructed
- 6 Loops of bowel may become intertwined and obstructed
- 7 Improper replacement of bowel within the peritoneal cavity may cause volvulus

An important physical finding in obstructions involving the anterior abdominal wall is localized pain and pin point tenderness at the site of small bowel adherence. This is caused by the peritoneal irritation produced by the obstructing process.

SPECIFIC SMALL BOWEL OBSTRUCTION FOLLOWING GASTRIC SURGERY

Obstructions in this group are the result of specific surgical procedures and are chiefly the result of the anatomic changes which the surgical procedure has produced. The three main types of surgery performed upon the stomach which can encourage the development of intestinal obstruction are the closure of a perforated ulcer, gastroenterostomy with or without vagotomy and gastric resection.

Obstruction after Closure of Perforated Ulcer of Stomach or Duodenum

Although intestinal obstruction is an uncommon sequel after the surgical treatment of a perforated gastric or duodenal ulcer, those obstructions which can result may be extremely lethal. Although the closure of a perforated ulcer might not seem on the surface to contribute to the production of small bowel obstruction, it is in fact the cause of the specific type of obstruction. The following etiologic mechanisms have been described:

- 1 A loop of small bowel may become adherent to the point of closure of the ulcer and thus become obstructed. This is especially apt to occur if long tails are left on the suture used in the closure.
- 2 If a live loosely attached omental graft is brought up and sutured over the point of perforation, a loop of bowel may slip through

the loop so formed and produce bowel obstruction.

- 3 The closure of a perforated ulcer by purse string or plicating suture may so narrow the duodenum or pylorus that stenosis and obstruction result.
- 4 A pelvic or subphrenic abscess may result from a perforated ulcer. This can cause three main types of bowel obstruction: (1) The abscess may produce intestinal obstruction by virtue of the edematous changes or plastic exudate upon the bowel in contact with it; or (2) The abscess may be large enough to cause bowel obstruction by compression; or (3) As a late sequel of an abscess, violin string adhesions may form which cause bowel obstruction years later.

Posterior wall ulcers may penetrate into the pancreas. As a result, bowel obstruction occurs. Two mechanisms are operative in the production of obstruction under these circumstances. These are: (1) a paralytic ileus resulting from the infection within the lesser peritoneal cavity into which the ulcer penetrated; and (2) a paralytic ileus occurring as a result of pancreatitis which is often caused by such penetrating ulcers. If a clinical diagnosis is made, the treatment should be conservative. In those cases in which a diagnosis is not made preoperatively, the gastrocolic omentum should be opened and the perforation closed. Those patients seen early by the surgeon may be subjected to definitive gastric surgery for the ulcer. Generally, cases of this type are best treated by suction applied to an indwelling gastric tube, intravenous alimentation, and the liberal use of the broad spectrum antibiotics.

Small Bowel Obstruction after Gastroenterostomy

The first gastroenterostomy was performed by Wolfer in 1881. Despite the fact that vomiting of a regurgitant type was a common complication and in many instances was so severe and persistent that it caused fatalities, the procedure became popular. The earlier gastroenterostomies were performed with a long proximal loop which was placed anterior to the transverse colon. Von Hacker believed that this was the cause of the

vomiting so often associated with this operation. For this reason he proposed making the anastomosis on the posterior surface of the stomach. The length of the loop however remained the same. This modification did not decrease the incidence of postoperative vomiting. Rockwitz believed that the failure of the first portion of the small bowel to function was due to a physiologic inability of peristalsis to pass from the stomach over the stoma and down the efferent loop of bowel. He suggested an isoperistaltic type of anastomosis as the logical method of correcting this. Braun and Jaboulay recognized the importance of postoperative vomiting and suggested the anastomosis of the efferent and afferent jejunal loops as an entero anastomosis. This did relieve the vomiting which followed the surgery. A great step forward was made when Peterson performed the first short loop posterior gastro enterostomy in 1900. This operation is still popular.

Experience in the past 10 years has demonstrated that the complication of intestinal obstruction at the stoma following gastro enterostomy cannot be explained entirely on a physiologic basis. Many cases are mechanically obstructed. The anastomosis does not function because it is obstructed. Such obstructions may occur regardless of whether the stoma is anterior or posterior. The length of the loop and its placement isoperistaltically or antiperistaltically do not influence the incidence of obstruction too much. Similarly the size of the anastomosis and its position on the wall of the stomach are not important factors in determining the development of postoperative obstruction.

It has been pointed out that there is a considerable variation in the normal anatomic relationships of the stomach, first limb of the jejunum and the transverse mesocolon depending upon the body type of the patient. In all cases obstruction after gastro enterostomy may be avoided by making the anastomosis in such a fashion that it will rest without tension when the patient assumes the erect position. As a result the site of the opening in the stomach and the length of the proximal loop are adjusted to make this possible. In addition the opening in the mesocolon must be made with due regard to the mechanics of the specific anatomic

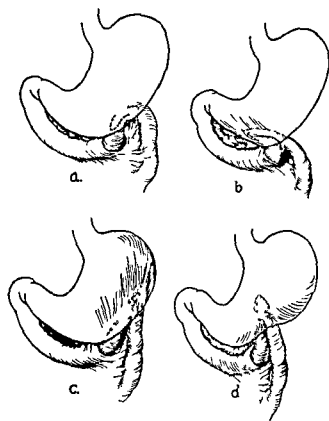


FIG 379 (a) Correctly placed stoma (b) Effect of stoma placed too far toward pyloric end of stomach (c) Effect of stoma placed too far toward cardiac end of stomach (d) Effect of stoma placed too high on posterior wall of stomach

problem. Its point may be anywhere near the base of the periphery or the right or left side depending upon the organ arrangement in the specific individual. Similarly the size of the anastomosis may vary widely. However an opening smaller than the lumen of the jejunum opening into it might cause obstruction. An opening of very large size is of little advantage since the stomach can only empty as fast as the lumen of the jejunum can accommodate its contents.

The first case in which obstruction occurred following gastro enterostomy was described by Kehr in 1899. This was only 18 years after the first gastro enterostomy was performed. Isolated case reports have appeared in the literature with sufficient regularity so that a classification based upon the mechanism of its production can be made. The following varieties have been reported.

1. Hellens and Nunn described the most uncommon type of small bowel obstruction following

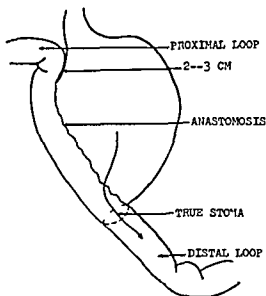


FIG 380 Post gastrectomy stomach. Note location of true stoma.

gastro-enterostomy. In this case some years after an anterior gastro-enterostomy with an entero-anastomosis between the loops it was found that the gastro-enterostomy stoma had completely closed. The intestinal loop had become detached from the stomach and had caused obstruction.

2 Prolapse of the jejunum through the anastomosis an uncommon cause of obstruction was reviewed by Aleman in 1948. He added two cases of his own to the 70 cases of acute or chronic obstruction of this type already reported. In obstructions of this type the process may be acute or chronic. In some cases the prolapse reduces itself spontaneously and such cases do not require surgical correction. In the acute type of prolapse surgical correction is indicated especially if the patient is in good condition. In such cases the gastro-enterostomy should be taken down and a partial gastrectomy performed.

3 If the opening in the mesocolon is made too small and if the stomach side of the anastomosis is not firmly anchored to it the anastomosis may be pulled up and become obstructed when the stomach fills. When the mesocolon is short, thick and fatty the opening must be made unusually large and the stomach drawn through it and firmly fixed in position to avoid this complication. Inadequate fixation of the gastric side of the anasto-

mosis to the circumference of the mesocolonic stoma may result in the development of a hiatus through which part of the anastomosis or a loop of small bowel may work its way and become obstructed. Keene, Armitage and Mayo have each reported cases in which this occurred. Adhesions may occur around the mesocolonic opening due to leakage or trauma. These adhesions may become progressively firmer and are a not uncommon cause of obstruction of the proximal or distal loop of the anastomosis. When the mesocolonic opening is made to the right of the middle colic artery the pressure of this vessel may obstruct the loop of bowel adjacent to it. A case of this type was reported by McCaughan and Coughlan. Marginal or jejunal ulcers may occur at or near the stoma with a resultant swelling and inflammatory reaction causing contracture and complete obstruction. After anterior gastro-enterostomy the mesocolonic opening is eliminated as an element causing bowel obstruction. The signs and symptoms of all the above types of intestinal obstruction are those of high intestinal obstruction. Prompt surgical correction is the treatment of choice.

4 An uncommon type of obstruction may occur following posterior gastro-enterostomy when the jejunum is anastomosed improperly to the posterior surface of the stomach on a horizontal plane. When the patient assumes the erect position the

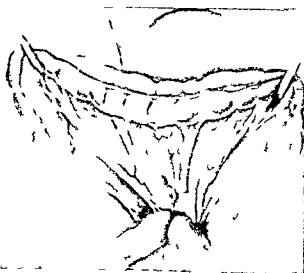


FIG 381 Obstruction of the proximal and distal loops as the result of being pulled through the transverse mesocolonic opening.

loop of jejunum tends to twist upon its long axis. As a result the proximal or distal loops may become obstructed at either stoma of the jejunum. This is especially apt to occur if the jejunum has become edematous and thickened. The signs and symptoms of intestinal obstruction appear in such cases within 10 days of surgery. It is imperative that the gastro enterostomy be taken down, the bowel resected and a new gastro enterostomy properly placed.

5. Anterior gastro enterostomy may be followed by obstructions of a different type. If the proximal loop is anastomosed to the greater curvature of the stomach as a short loop and the distal loop is anastomosed to the pyloric side in an isoperistaltic fashion and if the proximal loop is short, volvulus of the proximal loop is not at all likely. Retrograde intussusception of the distal loop may occur however through the anastomosis, producing bowel obstruction. Another type of obstruction may occur with this type of surgical procedure if a loop of small bowel or the distal loop of jejunum passes

into the space lying between the gastro enterostomy in front and the transverse colon behind. Such loops of bowel may slip into the space from left to right or from right to left. Volvulus of the small bowel so herniated occurs with a consequent early circulatory compression. McJiver reported a case of this type and Hublin reported several cases of internal herniation of small bowel with obstruction after posterior gastro enterostomy as well as after anterior gastro enterostomy. Steden reported a similar type of obstruction following anterior gastro enterostomy. In all of these the herniation had taken place in the same manner: a loop of small bowel had pushed into the hernial orifice from right to left or from left to right and the obstruction to the lumen of the small bowel soon became complete due to torsion of the intestine. The tendency toward early strangulation in all such cases makes immediate surgical correction imperative.

6. In those cases in which a long proximal loop is anastomosed to the pyloric side of the stomach

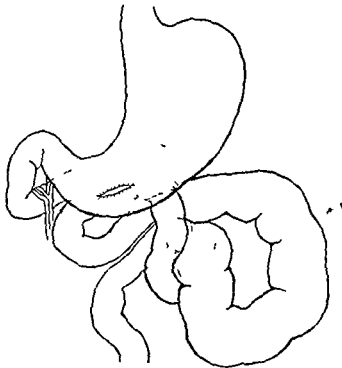


FIG. 382 Posterior gastro enterostomy followed by small bowel obstruction due to herniation of the distal loop of jejunum between the limbs of the loop formed by the proximal jejunal loop. This type of obstruction is the same as that reported by Hublin.



FIG. 383 Moderate dilatation of the duodenal loop with retroperistalsis secondary to stenosis of the afferent loop of a gastro enterostomy at the stomach.

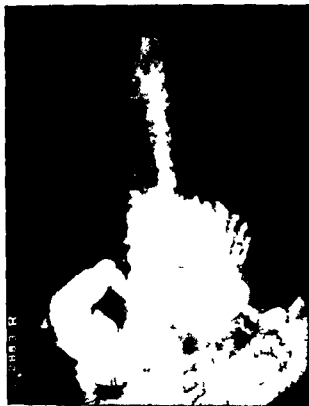


FIG. 384 Anther view of colitis as described in Figure 383.



FIG. 385 Malfunctioning gastro-enterostomy with stomal narrowing probably due to edema.

and the distal loop to the greater curvature side obstruction may result from the compression of the proximal loop by the mesentery if such mesentery is short and thick.

7 Stomal obstruction due to edema infection or kinking may occur at the orifices of the proximal or distal jejunal loops. If the proximal stoma is obstructed bile and pancreatic juice will regurgitate into the stomach through the pylorus if it is patent. If the pylorus is completely stenosed considerable duodenal distention may occur. In this event the line of anastomosis may be jeopardized by the distention of the wall. Serious consequences from proximal or distal jejunal loop obstructions at the stoma are uncommon because in most cases the pylorus is patent so that bile and pancreatic juice reflux into the stomach. From there they can be removed by suction via a Levin tube. Such is not the case however after subtotal gastrectomy since the duodenal stump is closed a closed loop obstruction occurs.

8 An important type of small bowel obstruction

as a result of gastro-enterostomy is axial rotation of the jejunum. This only occurs as a result of an error in surgical technique when the surgeon begins the jejunal anastomosis on the mesenteric edge and ends the anastomosis on the antimesenteric edge. As a result the jejunal loop anastomosed to the stomach becomes twisted on its long axis. Treatment consists of taking down the anastomosis and reconstructing it along straight lines.

Small Bowel Obstruction after Subtotal Gastrectomy

There are many types of intestinal obstruction after subtotal gastrectomy. The following varieties have been described.

1 Obstruction at the stoma. When we speak of the stoma following partial gastrectomy we do not refer to the entire gastrojejunal anastomotic line. We refer only to the lumen of the proximal or distal jejunal loop. Regardless of whether one performs a Polya or a Hofmeister type of anasto-

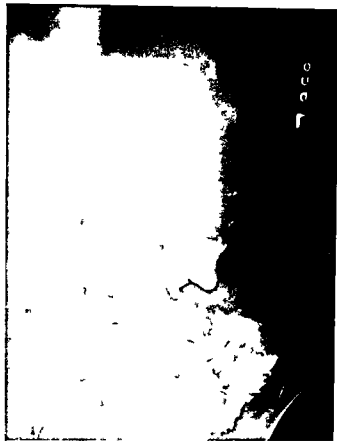


FIG 386 Structure of the efferent loop of a gastroenterostomy



FIG 387 Note that the true gastric stoma is the circumferential diameter of the distal jejunal loop into which the stomach opens

mosis the true gastric stoma is that opening into which the gastric contents are poured. This can only be the lumen of the jejunum that receives it.

There are six recognizable types of stomal obstruction. These are:

- a Stomal edema which may be the result of an electrolyte imbalance either hypochloremic or hypoproteinemic.
- b Infection at the line of anastomosis with localized abscess or inflammatory reaction.
- c Obstruction of the stoma as a result of adhesions or as a result of too high tacking of the jejunal loop to the lesser curvature of the stomach. This may result in a sharp angulation and obstruction as a result of the weight of the intestinal loop.
- d Paralysis of the distal jejunal loop. This is uncommon. It may occur when the pancreas has been traumatized during the release of a penetrating posterior wall ulcer. In four cases of this type reported by the authors

re operation was resorted to when conservative measures did not produce satisfactory results. At the second operation both the gastrojejunal and the stomal openings were found to be of normal caliber. In each case there was marked atony of the distal jejunal loop. In each case an entero anastomosis was performed and in each case the patient expired. It would appear from this experience that the treatment in such cases should consist of continued long tube intubation and tube feeding until the process producing the atony of the distal loop has resolved and peristaltic activity has been reestablished. Whenever possible jejunostomy should be avoided for feeding purposes.

- e A spur may be formed at the anastomotic line due to the descent of the efferent and afferent jejunal limbs which may cause obstruction.



FIG. 388 Specimen from experimental animal following gastrectomy. The anastomosis has been transected. Note the cuff of small bowel narrowing the circumferential diameter of the afferent and efferent stoma.

- f. As a result of a technical error too much tissue may be inverted in making the anastomosis. This may create a constricting ring within the stoma thus mechanically obstructing it.

When it has been established that stomal edema is the cause of the obstruction to the stoma the correction of the electrolyte imbalance and hypoproteinemia and the correction of the inflammatory reaction by the liberal use of antibiotics with decompression of the stomach usually result in a release of the obstruction. Plasma or whole blood may be given intravenously to correct hypoproteinemia. To correct protein deficiencies ground meat may be given by mouth via a fine plastic tube as suggested by Barron. The length of time required for this type of obstruction to relent is variable. The authors on one occasion treated a



FIG. 389 X-ray taken after a gastric resection. Notice the stoma of the distal jejunal loop. This is the circumferential diameter of the jejunum into which the stomach empties.

patient in this way for 23 days before the obstruction subsided and the continuity of the bowel was reestablished. Graham reported having treated a patient of this type for 36 days before the obstruction cleared up.

If radiologic study indicates that the obstruction is not due to stomal edema but is mechanical in nature surgical correction is needed. Lysis of adhesive bands is usually sufficient. In those cases in which a loop of bowel was tacked too high along the lesser curvature releasing this loop reduces the angulation and hence relieves the obstruction.

2. Obstruction of the distal jejunal loop may occur below the stoma as a result of adhesions to denuded areas of the pancreas. Narat and Manelli described a case of this type in which obstruction of the jejunum occurred 4 cm. below the anastomosis. The bowel became adherent to a denuded area of pancreas from which the stomach had been dissected free. In gastric resection when the

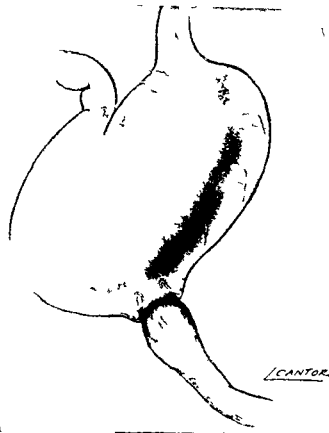


FIG 390 Drawing of post gastrectomy stomach. Here again note the true gastric stoma is merely the diameter of the jejunum into which the stomach empties.



FIG 391 Post gastrectomy stomach. Notice that the direction taken by the intestinal decompression tube is straight down. To successfully intubate this type of case the patient must be ambulated because the stoma is at the greater curvature which is the most dependent portion of the stomach.

posterior wall ulcer has penetrated into the pancreas by leaving the ulcerative lesion on the pancreas a large exposed area may become adherent to the adjacent afferent or efferent jejunal loop such as in the case reported by Narat and Manelli. This may cause cicatricial stenosis in the loop of bowel adherent to it. The treatment employed by Narat and Manelli consisted of an entero anastomosis between the segment of the jejunum of the efferent loop below the anastomosis to the anastomotic site. As a preventive measure it would be desirable to cover ulcerative open lesions of this type with an omental graft.

3 Volvulus of the stomach may occur at any time following subtotal gastrectomy. This is especially apt to occur if a long loop anterior gastrojejunostomy is used to reconstruct the bowel continuity. At times an adhesive band of omentum may wrap itself around the proximal and distal jejunal loops producing a mild partial obstruction. The resultant stagnation of intestinal content may

weight down the proximal and distal jejunal loops so that the stomach undergoes volvulus. In this event gangrene sets in quickly. Surgical detorsion as soon as possible is the only treatment. Volvulus of the stomach may occur in a different fashion. In a review of 425 subtotal gastrectomies at Grace Hospital volvulus of the stomach was found as a late complication in one case. The stomach had become adherent to the abdominal wall one third of the distance from the esophageal opening to the line of anastomosis. With this portion of the stomach acting as a fixed point the distal portion of the stomach and the gastrojejunum anastomosis had undergone volvulus producing a high intestinal obstruction.

4 Adhesions may cause small bowel obstruction after gastric resection by compression of either the gastrojejunum anastomosis or the proximal or distal jejunal loops. On occasion an omental sling

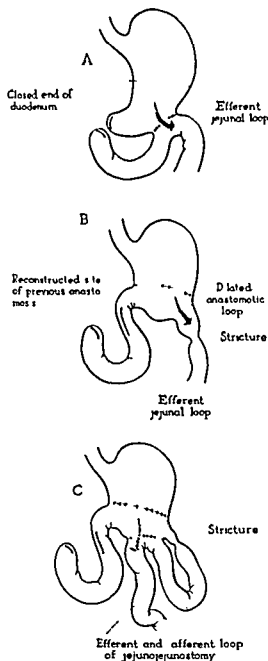


FIG. 392 Obstruction of efferent loop by stricture

may become adherent to the proximal or distal loops causing bowel compression. Another mechanism may occur in which the omentum may pass behind the gastrojejunal anastomosis and sweep from left to right over the proximal and distal jejunal loops obstructing them.

5. Internal herniation may occur following a gastric resection and cause small bowel obstruction.

In the case of a true Polya in which a posterior gastrojejunal anastomosis is performed the proximal or distal jejunal loops or both may herniate through the opening in the mesocolon. The herniated bowel invariably becomes obstructed. To prevent this complication the stomach should be well anchored to the opening in the mesocolon. If an anterior gastrojejunal anastomosis is performed the efferent jejunal loop may herniate into the recess behind the gastrojejunal anastomosis and the transverse colon in front. This herniation may occur either from left to right or from right to left. Strangulation of much of the herniated small bowel may occur as a result. Generally this opening is so wide that no real herniation capable of strangulating the bowel occurs. However in advanced cases this is apt to take place. Passage of intestinal contents through this herniated bowel is blocked so that a progressively increasing obstruction occurs. Stammers initially reported four cases of this rather uncommon complication in 1932. Since then other case reports have appeared. The number of reported cases however is still under 20. In the majority of the reported cases this complication arose during the early postoperative period, the earliest being on the third postoperative day and the latest on the eighteenth day. The average time of onset was the fifth or sixth day. In a smaller percentage of cases the obstruction did not occur until some time had elapsed after surgery, the earliest being $3\frac{1}{2}$ months and the latest being $2\frac{1}{2}$ years after gastrectomy. One case reported by Hofmann occurred 10 years after

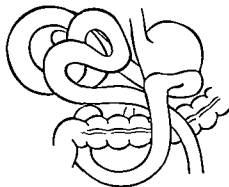


FIG. 393 Volvulus of the distal jejunal loop in which the loop has slipped into the recess between the transverse colon and anastomosis thus obstructing the distal loop at this point

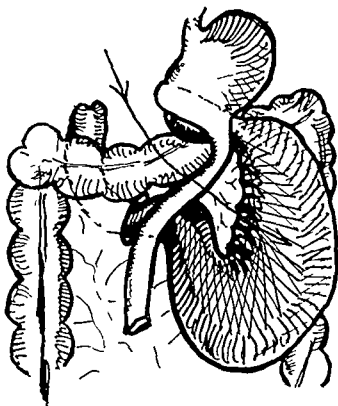


FIG. 394 Long afferent loop passing from right to left through the gap

gastric resection. The signs and symptoms associated with this complication are those of high intestinal obstruction. In the beginning the pain may last only an hour or so each day before becoming severe and shocking. Vomiting is not a prominent or essential feature at first and bowel movements may be normal for the first day. Distention is uncommon. The pain is generally reported in the upper abdomen and to the left. When gangrenous changes set in the signs of peritonitis appear. The management of this type of obstruction consists of early operation and reduction of the herniated bowel. The posterior surface of the stomach should then be sutured to the anterior surface of the transverse colon thus closing the gap. If gangrenous changes are present the involved loop should be resected and an end to end anastomosis performed.

6 Obstruction of the proximal loop is not uncommon. This is especially apt to occur if the gastroduodenal anastomosis is made anterior to the colon. Under such circumstances it is important to know whether the proximal loop is anastomosed

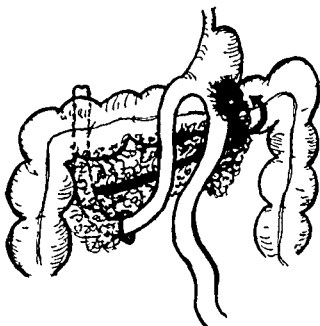


FIG. 395 The omentum has passed from right to left behind the anastomosis and become adherent to the front of the stomach and efferent loop

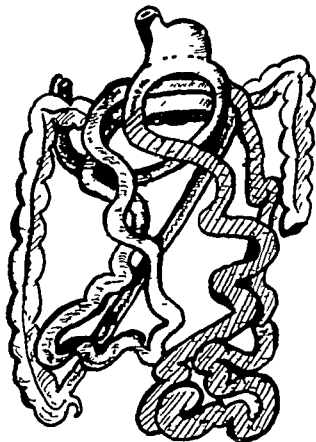


FIG. 396 In this case a loop of ileum (small intestine) from left to right through the gap (efferent loop) and curve

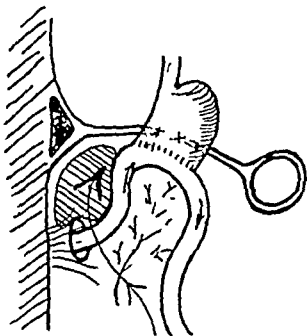


FIG. 377 A case of retrocolic gastrectomy in which the efferent loop passed from right to left through the gap

to the pyloric side of the gastrojejunostomy or to the greater curvature side. If the proximal loop was anastomosed to the pyloric side and the distal loop to the greater curvature side of the stomach two types of intestinal obstruction may occur:

- a. In this type the mesentery of the jejunum is short and as a result it may cut into or compress the proximal loop lying behind it. Proximal loop obstruction is produced by this process. Marked proximal loop distention sets in with the possible danger of duodenal stump blowout. Quinn and Gifford believe that this complication only occurs if an antecolic anastomosis is made between the stomach and proximal jejunum and if the proximal limb of the jejunum is anastomosed to the pyloric side of the stomach and the efferent limb anastomosed to the greater curvature side of the stomach. Many surgeons believe that as long as the jejunum is brought up and lies in the easiest and most natural position it does not matter whether the anastomosis is placed in one position or another. Quinn and Gifford believe that the direction in which the jejunum lies is extremely important. They point out that the

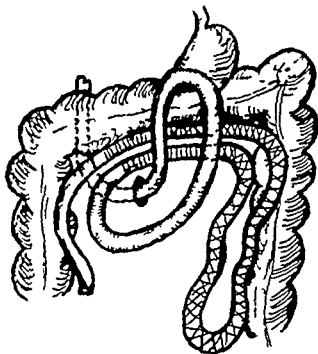


FIG. 378 Efferent loop passing from right to left through the gap and back into the greater sac again

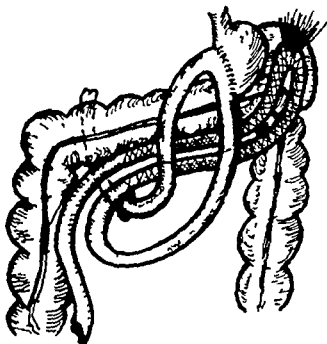


FIG. 379 Efferent loop passing from right to left through the gap becoming adherent to the spleen in the lesser sac

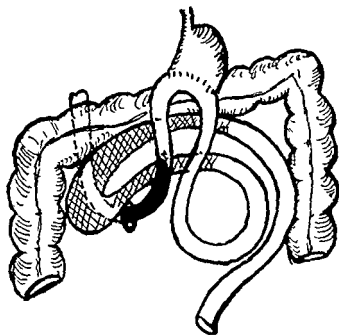


FIG 400 Showing efferent loop passing from left to right through the gap

mesentery lies in an oblique direction from the ligament of Treitz just to the left of the second lumbar vertebra and runs downward and to the right across the vertebral column ending opposite the right sacroiliac joint. When the jejunum is attached to the stomach so that the distal loop is at the greater curvature the mesentery of the distal loop necessarily becomes twisted approximately 135 degrees from this oblique line. Although the mesenteric torsion does not interfere with the blood supply of its own loop it does shorten the mesentery so that pressure is occasionally exerted upon the proximal loop as it passes between the shortened mesentery and the transverse colon. The pressure of the mesentery of the distal loop on the proximal loop may cause complete intestinal obstruction of the closed loop type. The continued secretion of bile and pancreatic juice into the closed loop causes marked distention of the duodenum and proximal jejunum. Perforation of the jejunum or duodenal stump blow out may result. The clinical picture in this type of obstruction is fairly typical. The onset is usually abrupt. Continuous and unrelenting epigastric or upper left quadrant

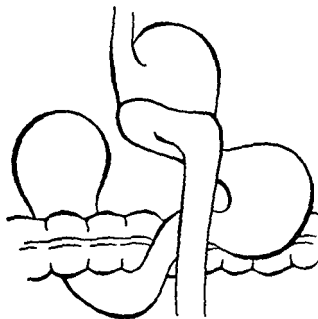


FIG 401 Intestinal obstruction as a result of a volvulus of the proximal jejunal loop in an anastomosis in which this loop is anastomosed to the lesser curvature of the stomach

pain and tenderness are noted and may be associated with shock of varying degree. The distended bowel may be palpable as a mass in the upper abdomen. Vomiting is usually not continuous, repeated or profuse and the vomitus does not contain bile or small bowel content. The character of the pain and the vomitus without bile are typical of this sort of obstruction. Radiologic study does not usually show any evidence of gas-filled distended small bowel loops. This is to be expected since no swallowed air can pass down the gastro intestinal tract and gas forming bacilli are absent in the proximal loop obstruction. Quinn and Gifford stress the fact that the prevention of this type of obstruction consists of simply anastomosing the proximal loop of jejunum to the greater curvature in the performance of the gastrojejunal anastomosis.

- b In this type of intestinal obstruction, the proximal loop may twist behind the anastomosis coming to lie between the transverse colon behind and the gastrojejunal anastomosis in front. A proximal loop volvulus results. In such cases the proximal loop lies

comes tremendously dilated and may become gangrenous because of strangulation or a duodenal stump blowout may occur. This also produces a proximal loop obstruction of the closed loop type. In cases of this type if no strangulation has occurred the proximal limb should be tacked to the distal limb with interrupted 000 silk sutures and an enteroanastomosis made between the loops. If gangrenous changes have occurred it may be necessary to resect the duodenal stump and the entire proximal limb transplanting the common bile duct and pancreatic duct into the jejunum. This is a formidable procedure in a patient in poor condition.

In addition to the specific types of proximal loop obstruction the proximal limb of jejunum may become obstructed by kinking at the anastomosis and also as a result of the development of a gastropyloric fistula.

7 Hublin reported a rather unusual type of bowel obstruction after gastrectomy and gastrojejunal anastomosis with enteroanastomosis between the proximal and distal loops. Volvulus of the small bowel occurred around the enteroanastomosis producing obstruction. In addition the small bowel may herniate between the limbs of the proximal and distal loops and thus become

obstructed. To avoid this complication the two limbs should be sutured together and also to the transverse colon behind with interrupted sutures.

8 Intussusception may occur as a result of herniation of either the proximal or distal jejunal loops through the gastrojejunal anastomosis. This is most likely to occur if the long proximal loop is present and is anastomosed to the pyloric side of the stomach. Retrograde invagination of the small bowel through the gastrojejunal anastomosis is occasionally found and has been noted after anterior gastrojejunostomy as well as posterior gastrojejunostomy. Tuomikoski collected 29 cases of this type from the literature and added one of his own. Up to 1951 the number of published cases of this type of obstruction did not exceed 50. In one case reported by Hublin this complication recurred two times after gastric resection. The amount of bowel intussuscepted retrogradely into the stomach is variable ranging from a few inches to as much as 2 feet. In most cases it may be easily reduced without necrosis. Occasionally necrosis of the intussuscepted loop may occur. This requires bowel resection. This retrograde intussusception may be acute or chronic. In many of the chronic cases the small bowel loop spontaneously reduces itself. In acute cases spontaneous reduction does not occur and surgical intervention is required. As a rule reduction is easily accomplished. Tacking the proximal and distal loops together is usually sufficient to correct this process. Intussusception of the proximal loop into the stomach may occur as late as 15 years after a gastric resection.

9 A rather rare and bizarre variety of intestinal obstruction is described as the vicious circle type. Hublin describes this as occurring after gastric resection or gastroenterostomy. In this type of case barium is seen to circulate from the stomach through the descending limb through the enteroanastomosis up the ascending limb and back into the stomach at a very rapid rate. Such cases become nutritional problems. Hublin suggests as treatment a resection of the ascending limb. This produces a Y shaped gastrojejunostomy. The mechanism involved in the production of this complication is ascribed to wrongly directed peristalsis which does not drive the stomach contents out into the intestine but instead through the

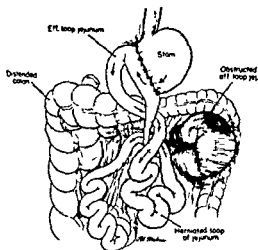


FIG. 40 The weight of the herniated distended partially obstructed jejunal loops has produced an even more severe secondary obstruction of the afferent loop of the gastrojejunal anastomosis. This in turn led to a perforation of this markedly distended bowel.

entero anastomosis and into the ascending limb and back into the stomach. Those cases in which an entero anastomosis was not performed in association with partial gastrectomy obviously would not develop this phenomenon.

10 A rare type of small bowel obstruction after subtotal gastrectomy is the type reported by Warner and Swan. This patient developed gallstone ileus $2\frac{1}{2}$ years after cholecystectomy for cholecystitis with cholelithiasis and $5\frac{1}{2}$ years after subtotal gastrectomy. The following mechanism was suggested by the authors. A small stone had entered the duodenum from the common bile duct prior to cholecystectomy. It had remained dormant in the duodenum for an unknown period of time gradually increasing in size. The previously existing subtotal gastrectomy with a functioning gastrojejunostomy would favor the growth of this stone if the proximal stoma were too small to permit its passage. The physiology of the first portion of the duodenum would simulate that of the biliary passage in the absence of gastric contents. The stoma of the proximal loop was adequate to permit the unobstructed passage of liquid duodenal contents but was too small to permit the passage of the stone. The stone increased in size until it eroded through the proximal loop of jejunum into the distal loop. The presence of a fistula between these loops furnished corroborative evidence for this mechanism. The stone then passed down the bowel and produced obstruction at the terminal ileum.

In general with regard to small bowel obstruction following gastric surgery it may be said that the nonspecific type of small bowel obstruction will probably always be a problem. Stomal edema may be prevented by proper preoperative preparation of the patient. Correction of electrolyte imbalance and protein deficits will reduce this problem to a minimum. All types of obstruction which are the result of a loop gastrojejunal anastomosis may be completely eliminated by the simple expedient of avoiding such loop operations whenever possible. The Billroth I procedure reestablishes the continuity of the bowel without a loop. In those cases in which a loop type of gastrojejunal anastomosis is necessary it would seem that the most desirable procedure would consist of anasto-

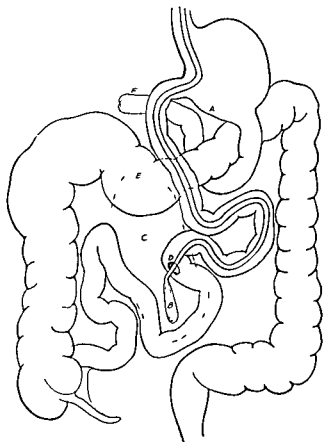


FIG 403 A Gastrojejunostomy B Intestinal decompression tube C Huge abscess D Perforation in jejunum E Perforation in colon F Duodenal stump This rare and hitherto unreported type of small bowel and colon obstruction followed a subtotal gastrectomy of the antecolic type performed four years previously. The transverse colon was obstructed by the effluent loop. As a result a perforation occurred in the transverse colon. This caused the development of a huge abscess. This in turn obstructed the jejunum. As a result of the jejunal obstruction a perforation appeared in the obstructed jejunum. This opened into the abscess cavity. A Cantor tube which had been passed to treat the intestinal distention was found at autopsy to have worked its way through the jejunal perforation. The end of the tube was found within the abscess cavity as noted in this illustration.

mosing the proximal loop to the greater curvature and the distal loop to the pyloric side of the stomach and then tacking the anastomosis to the transverse colon behind it. This obliterates the opening into this recess.

SPECIFIC SMALL BOWEL OBSTRUCTION AFTER COLONIC SURGERY

There are many types of bowel obstruction which can result from colon surgery. Mechanical

small bowel obstruction has been reported as occurring in 3 to 10 per cent of all cases in which some type of colonic surgery was performed. When colonic surgery requires a colostomy the incidence of complications has been reported to be between 25 to 30 per cent. In many cases more than one complication occurred with the colostomy. The types of obstruction following colon surgery may be divided into the following four large groups:

Obstruction Caused by the Pelvic Peritoneal Floor

Generally abdominoperineal resections carry approximately three times the number of small bowel obstructions as are found in anterior resections of the colon. This is due less to handling of the bowel than to the artificially created conditions associated with abdominoperineal resection since handling of the small bowel in the course of an anterior resection as well as in abdominoperineal resection is not appreciably different. Direct trauma does not appear to be a factor in the difference in the incidence of small bowel obstruction associated with these two surgical procedures. The three sources of small bowel obstruction to account for the differences are probably (1) the cut edge of the mesocolon (2) the suture line of the pelvic floor and (3) the aperture between the left pericolic gutter and the terminal colon as it ascends through the abdominal wall.

Adhesions between the small bowel and the pelvic peritoneal floor may be a source of intestinal obstruction. On many occasions such adhesions of the small bowel to the pelvic peritoneal floor may be the result of too long tails on the suture used to close the pelvic floor. On occasion a gap or rent may be left in the pelvic floor permitting a loop of bowel to slip through and thus cause obstruction. The obstruction produced in this fashion may be of a Richter's hernia type. A small portion of the antimesenteric surface of the small bowel may become strangulated in this fashion. This type of obstruction is particularly difficult to diagnose because the continuity of the small bowel is not interrupted. One of the first indications of its presence may be the development of a perforation and a pelvic abscess. The extensive suturing of

the pelvic floor and the presence of loops of bowel lying upon it present a favorable condition for adhesion formation and the possible development of small bowel obstruction. Obstruction of the terminal ileum may be caused by suturing of the pelvic floor to which the terminal ileum is attached by congenital bands or adhesions. Putting this flap on the stretch may cause a kinking and obstruction of the terminal ileum. To prevent this complication one must simply cut the congenital band at the time the pelvic floor is being repaired thus freeing the terminal ileum from its point of attachment.

Obstruction Originating in the Vicinity of the Colostomy

Obstruction of the small bowel may occur by becoming adherent to the peritoneal aspect of the colostomy wound or to the edge of the mesocolon which stretches towards the colostomy. This is responsible for a very small percentage of such small bowel obstructions. Intestinal obstruction may occur at times as a result of herniation of a loop of small bowel through the colostomy wound where it may become strangulated. These cases are by no means uncommon. Small bowel obstruction is rather infrequent as a complication of the colostomy itself without resection. The type of small bowel obstruction associated with colostomy is variable. There may be a herniation of the small bowel emerging through the opening in the abdominal wall through which the colostomy passes. The small bowel may pass between the lateral abdominal wall and the colostomy spur becoming obstructed in this fashion. On the other hand the small bowel may twist around with the colostomy spur as a central axis producing a volvulus. In those instances in which the colostomy is brought out through the midline the small bowel may herniate beneath the transverse portion of the colon sutured to the posterior abdominal wall and produce obstruction in this fashion. In addition a loop of small bowel may become adherent to the colon at the point at which it emerges through the peritoneum. Intestinal obstruction as a result of herniation of the small bowel through the peritoneal leaves of the iliac mesocolon has been reported.

Obstruction Originating in the Vicinity of the Anastomosis of the Colon

Obstruction of the colon may occur at the site of anastomosis in several different ways. If the colonic edges are turned in and too large a cuff is made as a result of an error in technique, obstruction of the colon may result. The development of infection in the suture line may produce a degree of edema in the turned cuff sufficient to effectively obstruct the bowel. This type of colon obstruction subsides with subsidence of the edema. Infection at the suture line of the anastomosis or leakage of the suture line produces a low grade peritonitis so that a paralytic ileus occurs. A leakage at the suture line may become walled off and an abscess may develop. Intestinal obstruction as a result of this abscess may be of several types: (1) as a result of the peritoneal irritation, a paralytic ileus may set in; (2) the formation of a pericolic abscess may cause adherence of a loop of small bowel to it with resultant angulation and mechanical small bowel obstruction; (3) edema of the small bowel wall may be of sufficient degree to mechanically obstruct it; (4) the abscess may reach such a size that it compresses the colon or the small bowel mechanically obstructing it.

Drainage of an abscess as a result of a leak at the suture line with the formation of a fistula may be responsible for the development of mechanical intestinal obstruction as a result of a loop of bowel becoming adherent to the fistulous tract. All these inflammatory types of mechanical intestinal obstruction are best treated conservatively by means of the intestinal decompression tube for the small bowel to correct the paralytic ileus and defunctionizing transverse colostomy to completely defunctionize the obstructed colon.

Obstruction at the Emerging Colostomy or Ileostomy

Stricture or stenosis about the emerging loop of colostomy is the most common complication. The narrowing in most cases is at the skin level and may be noted several weeks or a month post-operatively. This is more likely to occur in a single loop than in a double loop colostomy. Many surgeons believe that the stab wound alone is a

responsible factor because of an inadequate opening through the fascia of the abdominal wall. It may be difficult to decide at the time of surgery between an opening in the abdominal wall that may be snug and one that is too large so that prolapse or herniation of a loop of bowel may occur through the stab wound. In many cases the development of a wound infection is a predisposing factor to stenosis and as a result the fibrotic process associated with the healing of the wound may obstruct the colostomy. Ideally the colostomy should be flush with the skin and admit the tip of the index finger. Colostomy openings of smaller caliber than this may result in partial intestinal obstruction. Obstruction of the colostomy may also occur if there is too tight a closure of the deeper layers of the abdominal wall. Necrosis of the colostomy loop may occur occasionally in those cases in which there is impairment of the blood supply or in which the colostomy is brought out too tightly so that the mesenteric vessels or mesocolonic vessels become compressed and thrombosed. Necrosis may occur as a result of tension on the colostomy loop too tight closure of the abdominal opening through which the loop passes or improper division of the mesocolonic vessels. In an occasional case as a result of undue tension upon the colostomy loop it may drop back into the peritoneal cavity or may drop back for a variable distance beneath the skin producing an abdominal wall abscess. As a result of such accidents paralytic ileus or mechanical obstruction of the inflammatory distention type may occur. One of the most common complications associated with colostomy is prolapse of the colonic mucosa. This prolapse is usually not associated with mechanical intestinal obstruction. A large segment of bowel may protrude through the colostomy opening producing an intussusception. This is troublesome to the patient and may cause mucosal bleeding. In an occasional case sufficient mucosa may prolapse to mechanically obstruct the bowel.

In addition to the acute intestinal obstruction produced as a result of the colostomy the ileostomy syndrome has been considered as being due to a low grade obstruction of the ileum as it protrudes through the abdominal wall in any case.

requiring ileo tomy. Although the cause of this syndrome is not known the conclusion appears to be that it is a potential complication inherent in the establishment of any ileo tomy and may be due to an interference with the neuromuscular coordination of the bowel as it passes through the abdominal wall producing a functional type of intestinal obstruction. Turnbull has suggested that the ileostomy dysfunction producing obstruction following total colectomy and ileostomy may be due to edema of the pre existing limb. This is suggested by the fact that the enteric may respond promptly to intubation of the stoma and suction applied to it. It appears that the small bowel does not tolerate exteriorization over long periods of time.

Obstruction Caused by Blind Efferent Loop of Double Barrel Colostomy

An extremely unusual small bowel obstruction was that reported by Mackenzie. This patient had a perineal excision of the rectum nine years prior to the operation for acute intestinal obstruction. At surgery it was found that the colon distal to the previously performed colostomy was gangrenous. The gangrenous bowel proved to be the blind efferent loop of large bowel leading from the colostomy. It had not been fixed distally but had hung freely in the peritoneal cavity as a pendulous sac. At operation for obstruction the blind end of the colon was resected leaving a 1½ inch stump at the colostomy. The patient made an uneventful recovery. This rare and unusual complication can only occur when a distal segment of bowel is permitted to remain.

Diagnosis

A diagnosis of small bowel obstruction in association with surgery or following surgery of the colon may be very difficult to make. This is particularly true of those cases in which the obstructing process develops within the first four or five days following surgery. At this time the symptoms complained of by the patient are generally ascribed to gas pains as a result of the operative procedure or paralytic ileus which follows any cutting operation upon the small bowel or colon. In most cases

in which small bowel obstruction occurs following abdominalperineal resection or colon surgery of any type the diagnosis can be made if the surgeon considers its presence possible. In a small percentage of cases the diagnosis may be considered as probable but not certain and in a still smaller percentage of cases the diagnosis cannot be made or the indications are insufficient to justify surgical intervention. This latter group constitutes a small percentage of the cases in which small intestinal obstruction occurs after colonic surgery. Even in these cases a careful review of the problem from time to time throughout the first 48 hours of the onset of symptoms may result in an increase in the number of correct diagnoses.

In a review of the reported cases the critical period for small bowel obstruction following colonic surgery was found to occur between the sixth and the eighth postoperative days. The considerable mortality rate for this complication of colonic surgery is due to the fact that the correct diagnosis is not made or even suspected. In a large extent this is due to the understandable reluctance on the part of the surgeon to believe that his patient has developed small bowel obstruction in the first postoperative week. A prompt recovery is possible in those cases in which a correct diagnosis is made and immediate surgical intervention instituted.

The management of obstructions following major colonic surgery should in no way differ from the treatment of intestinal obstruction as a primary disease. In many cases exploratory operation is indicated even when a suspicion of intestinal obstruction is present and when intestinal intubation does not result in a prompt relief of all symptoms within the first 24 hours.

Partial wound dehiscence is a common cause of small bowel obstruction after colonic surgery. In many cases the wound separation involves only the deeper layers and the sutured skin remains intact. A serosanguinous drainage from the incision should be presumptive evidence that such separation of the incision has occurred. It is not unusual for a loop of small bowel to herniate through the opening made in the peritoneum and become obstructed. To prevent this in all sus-

pected cases of partial wound dehiscence the wound should be reopened and examined for herniated small bowel. Following this a secondary closure of the incision is in order. Many surgeons who would deal with a wound dehiscence promptly and adequately fail to recognize the fact that

moderate degrees of wound separation are far more dangerous to the patient than are the wide open incisions for which prompt surgical closure is mandatory. It is the hidden wound separation which cause most of the cases of small bowel obstruction from this source.

BACTERIA, TOXINS, AND ANTIBACTERIAL AGENTS IN BOWEL OBSTRUCTION

The beneficial effects of the antibacterial agents in the management of intestinal obstruction have been established suggesting that the intestinal bacteria and toxins play an important role in intestinal obstruction

ROLE OF BACTERIA

Bacteria have a very definite role in all cases of intestinal obstruction. In the presence of an obstructed bowel the bacterial count of the intestinal contents shows a marked increase. In an experimental study Laufman, Martin, Method, Tuell, and Harding demonstrated that a devascularized loop of intestine could be permitted to remain in the peritoneal cavity in the relative absence of pathogenic bacteria and that this strip of devascularized ileum would undergo almost complete autolysis with no harmful effects to the animal. This would only occur if the tissue were relatively free of bacteria. This suggests the important role played by bacteria in the production of toxic changes in the host. Harper and Blain, in their experimental work with dogs, observed that although the average survival time of a controlled animal was $3\frac{1}{2}$ days, all those animals in which penicillin was given parenterally or administered into the obstructed loops lived nine days and 60 per cent survived more than 30 days. As a result of this experimental work these authors concluded that by controlling the bacterial growth with penicillin or other antibiotics and by controlling the effects of intestinal distention the distention in the intestinal loop was not necessarily fatal in itself. It was also observed that the lower in the gastrointestinal tract the obstruction occurred the

greater was the degree of intestinal distention and concomitant with the increased intestinal distention the greater was the incidence of hemorrhage, necrosis, and ulceration. The use of adequate doses of antibiotics, largely penicillin, was found to prevent these mucosal changes. This suggested that although the distention may prepare the way for the bacteria to invade the bowel wall, bacterial growth may be responsible for the ulcerative, necrotic process occurring in the bowel wall.

Recent reports by Sarnoff and Poth and Sarnoff and Fine suggest that in addition to the vascular defects the bacteria present in the intestine may be a determining factor in survival from the effects of strangulation. They observed that although the ligation of all the venous return from a 20-cm segment of ileum was fatal to dogs, if the animals were treated for 10 days pre-operatively with Sulfasuxidine given orally, 70 per cent of the treated animals survived indefinitely. By preventing bacterial proliferation it was found that a collateral venous return would develop through the omentum, the mesentery, and into the adherent loops of bowel. After 30 days the bowel appeared essentially normal. Prior to this, an es has been hemorrhagic infiltration in the bowel wall is some early ulcerative changes of the wall, and that observed Blain and Kennedy, as by enterostomy, performed similar experiments, proof for and an antibiotic and they observed material in the mesentery survived 72 hours at which time to obtain. A demonstration that the devascularized is present in the fluid of four out of five and that the injection of Gaster and their associates in their death does

not establish conclusively that this toxic material can pass the barrier of live mucous membrane of the small intestine. Similar toxic substances can be demonstrated in normal intestinal tracts. However, it has been postulated that with an impairment in the viability of the intestinal mucosa like that occurring in strangulating obstructions or as a result of long standing intestinal distention associated with asphyxia, the transmural passage of toxic substances into the peritoneal cavity and absorption by this means may occur. This theory that toxemia was a major factor had to be abandoned as a result of the demonstrations by Hartwell and Hoquet and Gimble and Ross that the loss of fluid and electrolytes was the essential cause of death in high intestinal obstruction.

ANTIBACTERIAL AGENTS IN THE MANAGEMENT OF INTESTINAL OBSTRUCTION

The antibacterial agents effective in the gastro intestinal tract are classified as either bacteriostatic or bactericidal. The bacteriostatic agents are mainly the sulfonamide drugs. These drugs have definite disadvantages as intestinal antiseptic agents. They have a relatively narrow spectrum of effectiveness and being bacteriostatic can only inhibit the growth of organisms so that in order to remove the viable bacteria it is necessary to evacuate the contents of the bowel by mechanical cleansing of the mucosal surface. This procedure requires a considerable amount of time and does not become consistently effective in less than seven days.

Repeated experimental studies have demonstrated that the intestinal antiseptics are of great value because of their ability to alter the bacterial flora in the lumen of the bowel, reducing the bacterial invasion of the tissues of the bowel. With the reduction of the bacterial flora within the bowel and the consequent reduction of toxic material products, one generally is able to prevent thrombosis of the bowel wall blood vessels, particularly the terminal capillaries. In this fashion, by preserving the blood supply, one is able to protect the tissues of the bowel. There has been considerable evidence, both experimental and clinical, that the liberal use of these antibacterial agents has done much to lower the mortality rate in strangu-

lating types of intestinal obstruction since such agents prevent bacterial invasion which is one of the fundamental phenomena responsible for most local and some general disturbances associated with strangulating obstruction.

Bacteriostatic Drugs

These are the sulfonamide drugs. The sulfonamide drugs used in the management of bowel obstruction are Sulfasuxidine, Sulfathiazidine, and Sulfaguanidine.

Sulfathiazidine and Sulfasuxidine. Poth and McClure studied the administration of Sulfasuxidine or Sulfathiazidine in experimental animals subjected to both arterial and venous ligation. They were able to conclude from these experimental studies that the bacterial flora within the bowel were an important factor in bowel necrosis in intestinal obstruction and that Sulfasuxidine and Sulfathiazidine had a distinctly favorable effect in promoting the repair of damaged intestinal tissue. In numerous publications Poth and his associates demonstrated that the bacterial flora of the intestine could be changed appreciably by the oral administration of Sulfasuxidine and Sulfathiazidine. The effect of the administration of Sulfasuxidine and Sulfathiazidine on the survival rate of experimental animals following vascular damage of segments of ileum was studied and it was demonstrated that they had a markedly protective role by causing sterilization or diminution of the bacterial count of the bowel, thus reducing the incidence of necrosis and consequently perforation.

Some of the factors involved in the production of perforation of the bowel are:

1. Increased intraluminal pressure of a mechanical obstruction which tends to lower the capillary blood flow in the bowel wall.
2. Infection within the bowel. As the blood supply to the bowel wall diminishes, the bacteria within the lumen of the bowel invade the wall and there produce toxic substances which cause thrombosis of the arterioles and capillaries. This results in ischemia, tissue necrosis and finally perforation.

Sarnoff and Poth demonstrated by experiment

that with the administration of Sulfasuxidine 70 per cent of their obstructed animals recovered as compared with the 100 per cent mortality for a similar group not given sulfasuxidine. This gives some indication of the marked effect the sulfonamide drugs had in altering the bacterial flora within the bowel. They demonstrated that whereas normally ligation of the venous return from segments of ileum 50 cm. in length was always fatal when these experimental animals received therapeutic doses of Sulfasuxidine 70 per cent of them lived.

From the evidence presented one might conclude that by preventing the invasion of the bowel wall by bacteria the Sulfathaladine and Sulfasuxidine were of value in any strangulating obstruction or in any case in which intestinal distention is such as to decrease the capillary bowel circulation. In addition there is some evidence clinical as well as experimental that the alteration of the bacterial flora affected by the oral administration of these antibacterial agents favors the healing of bowel lesions whether they are surgically or traumatically induced.

Sulfaguanidine This sulfonamide is seldom used because of its toxicity when administered in large doses. It was the first sulfonamide specifically introduced for intestinal bacteriostasis. Although it is relatively nontoxic absorption from the intestinal tract occurs in sufficient quantity to require limitation of dosage and hence reduces its effectiveness. Repeated white blood cell counts are advisable during Sulfaguanidine therapy to detect a possible leukopenia.

Bactericidal Drugs

The bactericidal agents which include many antibiotics eliminate the organisms from the gastro intestinal tract much more rapidly than do the bacteriostatic agents. The antibiotics used in the sterilization of the gastro intestinal tract are streptomycin, penicillin, Aureomycin, Terramycin and Neomycin.

Streptomycin Streptomycin was the first of the antibiotics to be studied as an intestinal antiseptic. However it was found to be unsatisfactory because of the rapid development of resistant

strains of organisms. Ravdin and his associates studied the effectiveness of streptomycin as an intestinal antiseptic. Their experiments with oral streptomycin indicated that this agent was more effective than Sulfasuxidine in reducing fecal bacteria. However oral streptomycin is usually not absorbed in appreciable amounts from the gastrointestinal tract. It was shown that 95 to 98 per cent of the streptomycin administered in this fashion could be recovered in the feces where it exerted its beneficial action. Davis, Gaster, Marsh and Pritel did demonstrate however that streptomycin was effective in lowering the mortality rate of experimental rabbits subjected to strangulating passive obstruction. They showed that if a sufficiently large dose of streptomycin were given there were no deaths. Since they believed bacterial growth in the devascularized bowel wall was a major factor in perforation and gangrene of the bowel the essential mechanism underlying the prolongation of life in the treated rabbits in their experiments could be ascribed to the protective action of the streptomycin. On the basis of the results obtained in their studies it was suggested that in human beings the administration of streptomycin might be a useful adjunct in the treatment of strangulated intestinal obstruction particularly those cases due to acute arterial mesenteric thrombosis or embolism. In addition streptomycin should be given in intestinal obstruction when strangulation is suspected. It was also pointed out that the therapeutic value of streptomycin was dependent upon the length of the strangulated bowel. Since it was impossible to predict the extent of strangulation all workers on the subject agreed that streptomycin should be regarded only as an adjunct to and not a substitute for surgery. They believed that the action of streptomycin in preventing bacterial invasion into the ischemic area and thus maintaining anatomic integrity of the bowel permits time for an adequate collateral blood supply to develop. Since the streptomycin reaches the bowel wall through the blood vessels it necessarily follows that the more extensive the area of strangulation the less effective the streptomycin will be in preventing bacterial gangrene and perforation of the bowel wall. This means that the more extensive the strangulated loop is the shorter will be the

time available for the streptomycin to exert its beneficial effect

Aureomycin (Chlortetracycline) When Aureomycin became available it was used to sterilize the bowel. However as a result of his studies Poth found that it was not an acceptable antiseptic agent. Even though the flora were altered on occasion this effect was not consistently produced. It was also demonstrated that *Micrococcus pyogenes* and *Staphylococcus aureus* would frequently grow despite the Aureomycin. Rabinovich and Fine studied the effect of Aureomycin on the intestine subjected to vascular injury. Contrary to Poth, they found that the bacterial flora were markedly reduced when oral Aureomycin was used. Colm noted that after adequate pre operative preparation with oral Aureomycin and postoperative treatment with penicillin and intravenous Aureomycin the average survival time of dogs was twice as long as that of dogs not treated with these antibiotics. The bowel lumen was sterile at operation in dogs so treated. The histologic appearance of the strangulated bowel segment showed marked preservation of structural detail in contrast to the destruction in the animals not treated by antibiotics.

Morton, Furth, Hinshaw and Shilling studied the effect of Aureomycin in experimentally produced intestinal obstruction. The results obtained were inferior to those obtained when penicillin, streptomycin, Sulfasuxidine or Sulfathiazole were used. Because of its wide bacterial spectrum they believed that Aureomycin should be considered as having a place in the treatment of acute intestinal obstruction as an adjunct to proper and adequate surgical therapy. Morton and his co-workers also demonstrated that in order to be most effective the Aureomycin should be given intravenously.

Terramycin (Oxytetracycline) Terramycin was proposed as a wide spectrum intestinal antiseptic with bactericidal properties but it was found to present the same disadvantages as Aureomycin. With continued use it was found that even when Aureomycin and Terramycin were not used as intestinal antiseptics, nausea, vomiting and bloody diarrhea often developed. At times a choleraform type of disease would develop with a pseudomembranous colitis which was often

rapidly fatal. It has been recently demonstrated that this choleraform type of disease is due to an overgrowth of *Micrococcus pyogenes* on approximately the third or fourth day when the other organisms are diminishing so that a naturally occurring antagonism of the *Staphylococcus* was removed. Under normal conditions the *Staphylococcus* organisms within the bowel are usually present in small numbers. Shellenberger, Perry and Collett in studying the effect of various antibiotics as intestinal sterilizing agents found that postoperative complications caused by the use of the antibiotics occurred in 17 per cent of the cases. However in low intestinal obstruction Terramycin had a favorable influence on the mortality rate. Allen studied 48 patients treated with Terramycin noting specifically the effect of this antibiotic on the side effects of illness. None of the patients treated had received any antibiotic within 60 days prior to the study. Oral crystalline Terramycin hydrochloride capsules were used throughout the investigation. A dosage from one to four capsules (250 mg each) was given with water every six hours. Stool samples were examined before treatment was begun at the end of seven days treatment and after completion of 14 days treatment. The bacteriologic survey consisted of the determination of the total number of bacteria per gram by weight of sample stool and of the individual counts for anaerobes, aerobes, *Escherichia* species, *Lactobacilli* and yeasts. As a result of his study of these 48 patients Allen found that the oral administration of Terramycin did not produce antiseptic of the colon. There was an increase in the total bacterial numbers due to *Streptococci*, yeasts and *Proteus* species which occurred in many of the subjects studied. *Lactobacilli* decreased and the yeasts increased frequently after one week of therapy. Resistant strains of coliform and *Lactobacilli* appeared during the second week of treatment. In 65 per cent of the subjects treated some type of systemic side effects developed during this study. 33 per cent developed nausea, 35 per cent developed pruritus, 21 per cent complained of diarrhea and 19 per cent suffered abdominal cramps.

Penicillin Harper and Blain demonstrated that placing penicillin in isolated closed intestinal loops

protects animals to a considerable extent from the usual toxic manifestations. Blain also used penicillin to treat experimental animals with strangulated lower ileal loops of bowel. He found that animals treated with penicillin lived longer than did control animals without it. Laufman, Martin, Method, Tuell and Harding demonstrated as had Dragstedt previously that sterile autolysis of a dog's intestine was completely innocuous to the host if the continuity of the intestinal tract was maintained. They were able to reduce the bacterial content of devascularized loops of bowel sufficiently to permit the complete autolysis of appreciable lengths of intestine. This occurred without apparent harm to the animals studied. Penicillin could be depended upon if frank death of bowel tissue had not occurred. The penicillin could not be expected to have any effect on frankly necrotic tissue. The only function of penicillin was the sterilization of the bowel thus permitting sufficient time for the host to revascularize the loop. From these observations it was concluded that since autolysis of sterile nonviable tissue is nontoxic one should infer that the toxicity in strangulating obstruction is due entirely to bacteria. However the question as to whether the toxicity of autolysis in the presence of bacteria is a result of the action of bacteria, of bacterial toxins, either endotoxins or exotoxins, or of some other spreading factors in the presence of dead tissue is still controversial. It has been pointed out that when the bacteria have a substratum of dead or dying tissue upon which to act the combination results in the release of certain spreading factors. This contributes to a general state of local and generalized increased capillary permeability. As a result there is an increase in the toxic state of the animal. It has been pointed out in addition that the release of polypeptides from tissue autolysis is of no consequence unless bacteria are present in sufficient concentration or virulence. If these concepts are permitted it would seem that the toxicity of tissue autolysis is increased by the local invasion of the bacteria and that the toxicity of the local invasion of bacteria is increased by tissue autolysis. Therefore the integrity of the blood supply is equally as important as the bacterial invasion despite the fact that sterile tissue autolysis is harmless.

Blain, Kennedy, Cahillan and Harkins studied the effect of massive doses of penicillin in experimental intestinal obstructions. They found that with massive doses of penicillin experimental animals with strangulated bowels survived twice as long as untreated controls when in both groups shock, hemorrhage and electrolyte disturbances were adequately cared for. In addition the strangulated bowel was resected in five of the penicillin treated dogs with a complete recovery in four. As a result of their experiences in the laboratory Blain and his associates concluded that therapy with massive doses of penicillin should be recommended in cases of acute intestinal obstruction in which strangulation is believed to be present or in which strangulation cannot be ruled out. They stress the point however that this should be an adjunct to and not a substitute for early operation since penicillin by decreasing the bacterial flora within the lumen of the bowel acts only upon one of the factors involved in the high mortality rate of intestinal obstruction. In no case should the aggressive antibacterial therapy with penicillin which is indicated for all cases of strangulated intestinal obstruction be used as a sole method of treatment. Although their studies demonstrated that bacteria played an important role in the toxemia and death of the experimental animal with strangulated obstruction there were many other factors which also played important roles. Among these are hemorrhage, shock, loss of electrolytes, dehydration and intestinal distention. Although the experimental animal treated with penicillin survived for a much longer period of time than did the control animals, this prolongation of life was shown to have a definite limit at the end of which time the penicillin animals died. Blain and his associates were unable to determine whether this limit was set by the final inability of the penicillin in the dosage given to control the bacterial growth, whether there were fatal alterations in the metabolic processes in the body chemistry, whether the presence within the peritoneal cavity of necrotic strangulated bowel resulted in death or finally whether some combination of these factors was the responsible agent.

Neomycin. Neomycin was first used as an intestinal antiseptic in 1949. It was soon realized that

this antibiotic had strong bactericidal properties which caused the rapid elimination of bacteria from the intestinal tract. The response to a clinical trial was excellent. It was soon found that the yeasts grew out in large numbers immediately after the elimination of bacteria. In addition during the summer months an overgrowth of *Aerobacter aerogenes* also occurred. This was found to take place in approximately 10 per cent of the patients treated with neomycin. For this reason the value of neomycin as an intestinal antiseptic is somewhat impaired and Sulfasuxidine or Sulfathalidine must be given in conjunction with neomycin in order to inhibit the outgrowth of the *Aerobacter aerogenes*. This combination of drugs closely approaches the ideal requirement for an intestinal antiseptic. Neomycin has also been found to be capable of sterilizing the gastro intestinal tract in two or three hours when administered with castor oil. After being in contact with feces for 15 minutes in concentrations that are readily maintained in the gastro intestinal tract no viable bacteria could be isolated. This antibiotic however has limited activity against *Shigella* organisms.

Neomycin is fairly toxic when given parenterally but the amount absorbed when taken by mouth is so low that not a single toxic reaction was observed from its administration to 350 patients for periods as long as three months. In most cases neomycin will entirely eliminate bacteria from the gastro intestinal tract in 24 hours. Following this an overgrowth of *Monilia* may occur and the patient may show some of the manifestations of monilial involvement particularly the exudative patches that occur in the mouth and are known as thrush. Poth has found that a number of hemolytic as well as nonhemolytic *Streptococci* are fairly resistant to neomycin alone. Poth has suggested that the occasion may arise when it may be desirable to add bacitracin to the combination of neomycin and Sulfasuxidine for intestinal antiseptics.

Neomycin is relatively nonirritating. It has a low sensitivity index and appears to promote tissue healing. Clinically in man, the ingestion of 1 gram of neomycin every four hours for three days results in neomycin concentrations varying from a trace to 80 mcg per cc of blood serum. This is

considerably lower than the toxic blood concentration of 200 mcg per cc. Studies of the stool have demonstrated that neomycin concentrations in the feces indicate that the major portion of the antibiotic passes into the feces unaltered. The method of administration of neomycin in effecting intestinal antiseptics is important. The patient is given a low residue diet then a single dose of 2 oz of castor oil is given. One gram of neomycin sulfate is given immediately after the cathartic and is repeated every four hours for four doses. Following this one gram of neomycin is given every four hours. This pre operative treatment is usually carried out for 24 hours and should never extend beyond 72 hours. All attempts should be made to have an empty colon to insure the most efficient action from the neomycin.

DANGERS IN THE USE OF ANTIBACTERIAL AGENTS

The use of the antibiotic agents as well as all antibacterial agents in intestinal obstruction is not without danger. One of the greatest dangers is the tendency of some surgeons to depend upon the antibacterial agents and thus unnecessarily or unduly put off surgical procedures. It has been repeatedly emphasized by all workers that in no case should necessary surgery be unduly delayed during the time that the antibacterial agents are being administered. It has been repeatedly stressed that these antibacterial agents are simply adjuncts to the management of intestinal obstruction. In addition there is a high percentage of complications resulting from borderline survival of greatly damaged intestinal tissue. Among these are marked disturbances in the intestinal peristaltic gradient. This causes impairment of the normal function of that segment of bowel which had undergone strangulation. This often results in a physiologic stricture due to a physiologic interference with a peristaltic wave when it reaches the site of previous strangulation. In addition to this some of the experimental animals (treated with antiseptics for strangulating obstruction) actually presented a stenosis or a stricture at the site of the strangulation which caused partial bowel obstruction. In some animals there was a marked constriction ring at the point of occlusion which was due to an adhe-

sive blind. Advanced intestinal obstruction caused by these constriction rings was not an uncommon finding. In all of the experimental animals subjected to strangulating obstruction there was a massive formation of adhesions between the neighboring loops of bowel and the involved area. On several occasions the kinking of either the involved loops or the adherent loops was so marked that varying degrees of obstruction were present proximal to this area. Many cases of secondary intestinal obstruction occurred as a result of the plastic adhesions which followed the revascularization of the strangulated loop. These plastic adhesions are very likely to occur in the reduction of incarcerated hernias. Another possible complication is the development of intussusception. Intussusception may develop in the segments of bowel which had recovered from a strangulating obstruction. Although no report of this condition occurring in humans after strangulation has appeared, its occurrence in experimental animals was not uncommon.

For all these reasons and because it is very difficult at times to be certain that actual necrosis of the strangulated bowel has not occurred, all workers in this field are universally agreed that released strangulated loops of highly questionable viability should still be resected if the surgeon is at all concerned about the recoverability of the tissue. The role of the antibiotics in this event would be to keep the bacterial content of the affected bowel at a sufficiently low level to permit normal healing. It is important to point out that since the time element is a major factor in the management of intestinal obstruction, there may not be sufficient time to adequately sterilize the bowel before surgery. In addition, in those cases in which the intravenous broad spectrum antibiotics are given, strangulation of a loop of bowel does not permit the antibiotic to reach the point at which its effect is exerted because the blood supply to that portion of the gastrointestinal tract is obstructed.

EFFECT OF THE USE OF DRUGS IN BOWEL OBSTRUCTION

The advantageous use of drugs in the management of intestinal obstruction particularly in adynamic and spastic ileus is the result of the direct application of our acquired knowledge of their pharmacology and the physiology of their action. In former years shotgun prescriptions of various substances most of which were foul smelling and many of which were of doubtful origin were used in the treatment of intestinal obstruction. During the 17th, 18th and 19th centuries every conceivable organic material was given by mouth in the treatment of ileus. Sydenham made a great contribution to the medicine of his era by discarding all of these concoctions. Although the incidence of cure in intestinal obstruction was not increased thereby, at least his patients were made more comfortable.

DRUGS THAT STIMULATE INTESTINAL ACTIVITY

Therapeutically, drugs that stimulate intestinal activity in those cases of intestinal distention unassociated with mechanical obstruction produce their effect in one or more ways:

1. Drugs that sustain the acetylcholine metabolism in the intestine by supporting those mechanisms which have been shown to be necessary for acetylcholine metabolism.
2. Anticholinesterase drugs which increase the action of the acetylcholine produced by the body.
3. Stable parasympathomimetic compounds such as mechoyl which may be used in the absence of acetylcholine formed by the body.

4. Sympatheticolytic drugs which either eliminate the sympathetic adrenergic nerve impulses to the intestine or interrupt the nervous reflex arc. In the former class ergotamine tartrate is good whereas in the latter class splanchnic or spinal anesthesia is effective.
5. Drugs that remove the stimulus which increases the inhibitory intestinal reflex.
6. Hormones, vitamins, and electrolytes which correct deficiency states.

There is adequate evidence to indicate that the excitatory action on the intestinal tonus and motility is due to the production of acetylcholine. Factors which produce atony or paralysis of the intestine do so as a result of either inducing a diminution of the intestinal content of acetylcholine or by so increasing the threshold of nerve impulse required to produce intestinal activity that the normal quantity of acetylcholine present is inadequate. Any decrease in the amount of acetylcholine present in the bowel wall therefore results in some degree of atony of the bowel. In addition to this reflex stimulation of the sympathetic inhibitory nerves to the intestinal wall or stimulation of the adrenal results in a relative decrease in acetylcholine effect because of the inhibition produced. As a result the intestinal tonus and motility conceivably may be increased by decreasing the inhibitory nerve impulses and the adrenal stimulation as well as by stimulating the production of acetylcholine directly. The elimination of the overacting sympathetic nerve impulses to the intestine may be accomplished by the use of the

sympathetic drugs such as ergotamine tartrate by sympathetic block or by spinal anesthesia.

The use of acetylcholine by injection to stimulate peristaltic activity is not desirable. When given by injection the acetylcholine is rapidly inactivated by cholinesterase. In addition to this there may be dangerous and unpleasant side effects with doses of acetylcholine sufficiently large to produce intestinal motility changes.

Satisfactory substitutes for acetylcholine are available in the anticholinesterase drugs. These are particularly valuable in enhancing the effect of acetylcholine in those cases in which there is a normal or adequate production of acetylcholine. However, if there is no acetylcholine production by the body, there will be little or no effect from the anticholinesterase drugs. In cases of this type, Urecholine is especially useful. This drug is one of a group of choline esters which act primarily as stimulants of the parasympathetic system. The effect of many of the cholinergic or parasympathomimetic drugs is believed to be due to inhibition or inactivation of cholinesterase. This results in the conservation or accumulation of acetylcholine in the tissues. With respect to the gastrointestinal tract, the effect of such cholinergic drugs is to improve the tonus and motility of the smooth muscle. Stigmonene bromide and Prostigmin are excellent drugs in this class.

Mecholyl is a stable parasympathomimetic compound which is more stable than acetylcholine and its effect upon intestinal tonus and motility is more favorable even in those cases in which Prostigmin is ineffective. The contra-indication to the use of Mecholyl is the danger of cardiac arrhythmia.

Ergotamine is described as a sympathetically acting drug. The primary action of this drug is to block the sympathetic inhibitory impulses. This enhances the parasympathetic activity. Splanchnic block effectively increases intestinal tonus and motility by blocking the inhibitory sympathetic impulses. Spinal anesthesia has a similar mode of action.

The removal of the stimulus which increases the inhibitory sympathetic activity will effectively increase the intestinal activity. The method used to accomplish this depends upon the etiologic factor producing the sympathetic stimulation. This varies greatly from peritonitis to retroperitoneal hemor-

rhage caused by a leaking aneurysm. It is obvious that this phase of treatment must be highly individualized.

It has long been known that thiamin deficiency impairs intestinal tone and motor activity. Hublin noted that a definite reduction in muscle tone was the outstanding feature of vitamin B deficiency in dogs. In addition, there was a marked delay in gastric emptying and definite small bowel hypomotility and colonic stasis. Similar changes were reported by Elsom and Drossner who also noticed an increase in the caliber of the jejunal loops in the experimentally produced vitamin B complex deficiency states in humans. Kantor believed that the gas and fluid levels which appear on the survey films of the abdomen of patients suffering from vitamin B deficiencies are suggestive of ileus. Correction of the vitamin B deficiency is almost immediately associated with increased peristaltic activity. Changes in the small intestinal motility due to vitamin deficiency states have been noted by Golden. Similar motility disturbances were noted in hypoproteinemia and in the intestinal tract of the newborn with its undeveloped nervous control. Golden suggests that in such cases there may be a submucosal edema which interferes with the nutrition and function of the intramural nerve cells in the intestine. Mackie has noted degeneration of the nerve cells in the intrinsic nerve plexuses of the intestine in deficiency states, lending some corroboration to the suggestion of Golden.

Postoperative nutritional disturbances are often associated with impaired intestinal motility due to the lowered osmotic pressures below the edema level. The marked ileus not infrequently associated with uremia has also been noted on the same basis. In fact, many clinical disorders associated with states of edema almost invariably carry with them some degree of ileus. In such cases, correction of the edema almost invariably is associated with symptomatic improvement.

In addition to the edema as a result of the previously mentioned disorders, ileus may result from edema of the intestinal wall due to overhydration and the excessive use of sodium chloride. Because of the widespread use of saline solutions in the pre- and postoperative state, this is not an uncommon occurrence.

Although the importance of ascorbic acid has not yet been established Harg and Tahirferro investigated the effect of ascorbic acid on intestinal activity. *In vitro* studies of intestinal muscle in the guinea pig showed an increase in tonus with a high concentration of ascorbic acid. Forster directly applied this knowledge by combining ascorbic acid and thiamin in the treatment of ileus with excellent results.

DRUGS THAT DEPRESS INTESTINAL ACTIVITY

Studies by Bisgard and Johnson have indicated that the barbiturate drugs depress gastro intestinal tonus and motility. The barbituric acid derivatives apparently produce varying degrees of anoxia due to respiratory and circulatory depression. In addition they cause an inhibition of tissue oxidation. It is believed by some workers that histotoxic anoxia is the most important factor within the therapeutic range of the barbituric acid drugs.

Chapman Rowlands and Jones studied the effect of atropine upon the motility of the upper small bowel. They noted a definite decrease in propulsive and total contractions in the bowel. There was a marked variation in motility from person to person and in a given subject from hour to hour. Even using placebos a 30 per cent decrease in intestinal contractions occurred.

Chapman French Hoffman and Jones studied the effect of Banthine, belladonna and placebos on upper intestinal motility by using a multiple balloon kymograph recording in the gastro intestinal tract. They demonstrated that Banthine caused a striking decrease of propulsive and total contractions and a slight to moderate decrease in tonus. The Banthine acted more rapidly and inhibited motility to a greater degree than did tincture of belladonna. Both drugs produced a significantly greater effect than that observed after placebo administration. The effect of Banthine and belladonna and their inhibitory action on intestinal motility may be satisfactorily applied in the management of spastic ileus or the treatment of Ogilvie's disease (persistent spastic contraction of the colon) producing intestinal obstruction.

The drugs used to induce anesthesia vary in their action upon the gastro intestinal tract. Bisgard and Johnson suggest that the influence an

individual anesthetic drug has upon the bowel depends upon its influence on the relative quantity of oxygen in the blood or tissues during the anesthesia. A decrease in tissue oxygenation results in decreased motility whereas an increase in oxygenation results in an increase in intestinal motility under such conditions.

EFFECT OF DRUGS ON INTUBATION

In reviewing over 500 intubations in the two year period from 1949 through 1950 studied from the point of view of the effectiveness of drugs upon the process of intestinal intubation three fairly well defined groups of cases emerged. These groups are:

- 1 Patients with atony of the bowel due to old age or paralytic ileus
- 2 The nervous high strung apprehensive patient who becomes panicky at the mere sight of a long intestinal tube
- 3 Patients with pylorospasm so that successful passage through the pylorus could not take place despite correct positioning with the tube head shown by X ray to be at the pylorus

Group 1

This group consisted of all those patients intubated for atony of the small bowel or paralytic ileus. Abdominal auscultation revealed very weak and infrequent intestinal sounds. In many cases an almost entirely silent abdomen presented itself. Elderly patients with poor intestinal propulsive activity and all those patients presenting the silent abdomen of paralytic ileus secondary to peritoneal inflammation were found in this group.

In cases found in this group the use of Prostigmin and Urecholine was found to be very effective. Because of their effect upon the small bowel both of these drugs were found to speed up the downward progress of an intestinal decompression tube which had already passed through the pylorus. Urecholine was especially useful in the elderly patients presenting a gastric as well as an intestinal atony. Figures 404, 405 and 406 present evidence of the effectiveness of Urecholine in this type of case. Since it stimulates gastric motility and increases gastric tonus this drug is quite effective in the treatment of an atonic stomach.

Case Report

A 72 year old woman was admitted to the hospital with a tremendous intestinal distention. Carcinoma of the pelvic colon was found to completely obstruct the bowel. Despite the complete obstruction hyperperistalsis and borborygmus were absent. The patient's musculature was weak and flabby. Her abdomen was almost silent on auscultation only an occasional tinkle being audible. A Cantor tube was passed with ease. Twenty four hours later a survey film of her abdomen showed the tube head to be in the stomach. Injections of 5 mg. of Urecholine every four hours were begun. A survey film of the abdomen 24 hours later showed the tube head well down the small bowel and 48 hours after intubation the tube head was found in the ascending colon.

Group 2

These patients are anxious and apprehensive. They become uncooperative when one attempts to pass an intestinal tube. Morphine and atropine were found to be effective in intubating individuals in this group. We believe that the effectiveness of these drugs depends chiefly upon the feeling of



Fig 40b The same patient as in Figure 40a. The first tube was removed and a second tube containing 9 cc. of mercury was passed. This film was taken 24 hours after the passage of the tube with 9 cc. of mercury.



Fig 40a The balloon containing 5 cc. of mercury remaining in the stomach of a 72-year-old woman with atonic musculature. Urecholine given. Tube removed and 9 cc. mercury placed in fall.

well being which the morphine produces and upon the antispasmodic action of the atropine. Chapman, Rowlands and Jones studied the effect of injection of 10 mg. of morphine upon the motility of the upper small bowel in eight adult patients. They found that while morphine caused a sustained decrease in propulsive contractions it also caused frequent spasms in the upper small bowel. The use of atropine with the morphine serves to abolish the spasms induced in the duodenum and upper small bowel by the morphine.

That the effect of these drugs was chiefly due to the action of the morphine in producing a mild euphoria is further suggested by the observations of Follev and Abbott. They concluded as a result of their studies that neither of these drugs had any effect upon gastric emptying. Since intestinal motility is decreased by morphine and atropine in intubating patients in this group if we were depending upon the effect of morphine and atropine on intestinal motility we could expect to encounter



FIG 406 The same patient as in Figures 404 and 405 X ray taken 48 hours after the passage of the tube containing 9 cc of mercury. Notice the presence of the tube head in the ascending colon

considerable difficulty. The fact that intubation was rapidly and successfully accomplished after the use of morphine and atropine would suggest that this was not due to the action of the drugs on the bowel *per se*.

The effectiveness of morphine and atropine in these nervous apprehensive individuals has been proved clinically. On 10 specific occasions routine intubation failed. These patients were then given 10 mg. of morphine sulfate and 0.45 mg. of atropine sulfate. Successful intubation resulted in each instance. The following case report is typical of this group of patients.

Case Report

T. K., a 42 year old female, was admitted to the hospital with a diagnosis of ruptured appendicitis. Survey film of her abdomen showed a marked small bowel distention. She was highly nervous and apprehensive. A Cantor tube was passed through her nose with some difficulty because she was uncooperative. The tube coiled in the lower esophagus as shown in Figure 407. This tube was removed and an appendectomy performed under nitrous oxygen

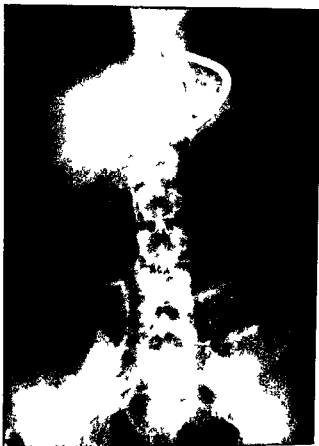


FIG 407 The effect of cardiospasm in the nervous apprehensive patients. Notice the balloon of the long tube trapped in the esophagus.

ether anesthesia. After she had recovered from the anesthesia she was given an injection of morphine and atropine and the Cantor tube passed into the stomach with ease. In 18 hours a survey film of the abdomen showed the tube to be well down the small bowel. The only change in technique in the first and second attempt at intubation was the use of morphine and atropine to allay nervousness.

Group 3

This group is composed of those patients in whom the tube head would not pass through the pylorus despite correct positioning as shown fluoroscopically or radiographically. These patients were classified as having pylorospasm. Stimulated by the report of Filho who noted that Octin relaxed the pyloric sphincter permitting successful intubation in 30 minutes or less in 90 per cent of cases, we tested the effect of this drug upon patients in this group. Octin is an antispasmodic which is said to relieve spasm by direct relaxation of the smooth



FIG 408 The same patient as shown in Figure 407 following the administration of morphine and atropine to allay nervousness and spasm

muscle. It is also said to stimulate the inhibitory fibers of the sympathetic system thereby enhancing the antispasmodic effect upon the pylorus. It acts quickly and its effect is prolonged. The antispasmodic action of the Octin usually begins two to four minutes after an intramuscular injection. Octin methyliso-octenylamine is an unsaturated aliphatic amine. It is available as Octin mucate in the form of a white powder with a bitter taste which may be given orally in a dose of 0.12 gm and is also available as Octin hydrochloride in solution for injection only. In this latter form the usual dose is 0.1 gm.

In the first 10 patients intubated using Octin hydrochloride only two patients presented pyloric spasm and a hindrance to successful intubation. In both attempts to pass the long tube were unsuccessful for one and two days respectively. However, successful intubation occurred in both patients within three hours after the injection of

the Octin hydrochloride. In 5 of the 10 patients intubation was unsuccessful within a 24 hour period. Three of these five were completely obstructed and had not been operated upon prior to intubation. Two of the five failures were patients who had been operated upon one to two days previously. Five of the 10 patients consecutively intubated were found to have successfully passed the tube through the pylorus in one to two hours after the administration of Octin hydrochloride. Of these five two patients were intubated for postoperative ileus. In both of these patients attempts to pass the tube by the routine method failed. These presented clinical evidence of pyloric spasm (see Figure 409). Of the five patients one presenting an acute abdomen was successfully intubated with the Octin. The remaining two patients successfully intubated had a long tube passed



FIG 409 Successful passage of tube through the pylorus as a result of the use of Octin. The passage of a tube through the pylorus was previously unsuccessful because of pyloric spasm.

as a pre operative measure prior to elective bowel surgery

From the foregoing experience as well as our subsequent experience it would seem that the use of Octin hydrochloride is of considerable value in cases presenting signs of pylorospasm. The Octin was of very little value in patients without pylorospasm however and should never be used in patients with paralytic ileus if the pylorus is atonic. However when properly used Octin has proved to be the deciding factor in producing a successful intubation.

THE EFFECT OF DRUGS ON ILEUS

For many years morphine was used in the treatment of peritonitis with its associated paralytic ileus. Although the rationale in its use was not clearly understood there is little doubt that satisfactory results were obtained. Morphine was first used clinically in the treatment of peritonitis by Graves in 1821. However its use became popularized by the publications of Alonzo Clark in 1879.

It was formerly believed that morphine decreased intestinal motility and produced a splinting effect upon the bowel which permitted healing to occur. We now know that this is not true. Recent experimental studies have shown that morphine promotes intestinal tonus and motility which is probably the reason for its beneficial effect in the treatment of peritonitis. Chesterman and Sheehan in a study of the prophylactic effect of morphine on paralytic ileus noted that in the group of patients not given morphine paralytic ileus occurred in approximately 5 per cent of cases whereas in the group given morphine prophylactically following surgery paralytic ileus occurred in only 0.88 per cent of cases. They therefore believed that morphine was of definite value in the prevention of postoperative paralytic ileus. The experimental studies of Ochsner, Gage and Cutting with paralytic ileus superimposed upon a mechanical obstruction demonstrated that the administration of morphine caused an actual increase in intestinal motility in 93 per cent of the cases studied and that in no case was there a decrease in activity. Similar results were obtained with both normal and obstructed intestines by many other observers.



FIG 410 Notice balloon of long tube trapped in the lower esophagus. The patient is suffering from paralytic ileus as a result of a ruptured appendix.

Clinically it can be demonstrated that the subcutaneous injection of morphine increases intestinal peristaltic activity. For these reasons Ochsner believes that morphine should be used and that it is a valuable drug in the prevention and treatment of adynamic or paralytic ileus.

The final role of morphine has not been established. There still appears to be some controversy over the use of this drug. Bernheim and Bernheim have shown that morphine inhibits the cholinesterase. The increased tonus noted clinically in the intestinal tract after morphine administration is considered to be largely dependent upon its anticholinesterase properties in the bowel wall. However it has been shown that intestinal tonus and nonpropulsive motility are increased after the administration of morphine. There is also some divergence of opinion as to the value of morphine in the prevention and treatment of postoperative gastrointestinal intubation states. Although there are many who believe that morphine is of great value



FIG. 411 The same patient as in Figure 410. The tube has passed through the cardiac sphincter of the stomach and has come to lie in the fundus of the stomach as a result of administering $\frac{1}{8}$ gr. of morphine sulfate and $\frac{1}{400}$ gr. of atropine sulfate.



FIG. 412 The same patient as in Figure 410 and 411. It is now evident that the tube has passed beyond the duodenojejunal flexure and will successfully decompress the patient.

in the treatment of postoperative paralytic ileus. Adler, Atkinson, Ivy, and Ingelfinger, among others, believe that unless morphine is needed for its analgesic action, it should not be used in the treatment of paralytic ileus or of any condition in which there is impairment of intestinal transport, since morphine may cause dyskinesia of the small and large bowel so that actual propulsion of intestinal contents is impaired.

Although the importance of thiamin deficiency in the production of ileus has been demonstrated, nevertheless no vitamin or group of vitamins directly stimulates or inhibits intestinal motility when a deficiency state for that specific vitamin does not exist. Glick and Antopol have demonstrated that vitamin B₁₂ inhibits the cholinesterase *in vivo* and *in vitro* and encourages or increases the physiologic action of acetylcholine. Banerji studied the use of calcium pantothenate in para-

lytic ileus and reported uniformly good results in the treatment of 19 patients.

It has been demonstrated by many workers that hypertonic saline solution stimulates the intestinal tonus and motility. Hypertonic sodium chloride solution, however, increases the intraluminal intestinal pressure and as a result its use should be contraindicated if there is unrelieved mechanical obstruction. It is of value in the treatment of paralytic ileus, especially the type of ileus associated with decreased sodium or chloride. On the basis of experimental and clinical studies, Ochsner believes that the intravenous administration of solutions containing calcium and potassium chloride are more efficacious in stimulating bowel activity than are solutions containing sodium chloride alone.

A wide variety of drugs have been used in an attempt to stimulate the motor activity of the intestine in paralytic ileus. These are as follows:

Eserine This drug has a stimulating action upon the parasympathetic system. It causes increased intestinal peristalsis. It also causes bradycardia, dyspnea, myosis and a drop in blood pressure. Because of these latter effects Eserine does not lend itself well to the treatment of paralytic ileus.

Prostigmin This is a synthetic compound resembling physostigmine in some respects although it differs from it chemically in that it has a less complicated structure and greater stability. It has a pronounced effect upon peristalsis, a less pronounced myotic effect and produces almost no cardiac side effects. The efficiency of Prostigmin as a peristaltic stimulant has been demonstrated unequivocally. It is generally administered in a 1:4000 solution of the dimethylcarbamate ester of 3-hydroxyphenyl ammonium methyl sulfate. This has been found to be satisfactory for the prevention or treatment of the distention of postoperative paralytic ileus. Considerable literature has accumulated on its use both pre-operatively and postoperatively in the prevention and treatment of paralytic ileus.

Pituitrin This drug takes its most marked action on the colon and its effect upon the small bowel may actually be to decrease intestinal activity. In those cases in which rapid evacuation occurs following the administration of Pituitrin extract the action of Pituitrin probably has been largely on the colon. Since in paralytic ileus the small bowel as well as the colon is involved in the atonic process it would seem that Pituitrin would be of little or no value in the treatment of this type of disorder.

Prostigmin methyl sulfate and thiamin hydrochloride Stout has proposed the routine use of Prostigmin methyl sulfate and thiamin hydrochloride given hypodermically every four hours postoperatively to prevent postoperative paralytic ileus.

Stigmonene bromide This is a new cholinergic compound which has been developed recently. Its use is indicated wherever a

parasympathomimetic drug is needed to combat intestinal distention. The dosage for postoperative abdominal distention or atony is 1 cc (0.5 mg) by intramuscular injection immediately following conclusion of surgery. The intramuscular injection of 0.5 mg every three to four hours is to be repeated until a total of six injections are given. Stigmonene bromide as well as all true parasympathomimetic drugs should never be used in any case in which there is a possibility of mechanical intestinal obstruction. A specific antidote in the event of an overdose of Stigmonene bromide is $\frac{1}{100}$ gr atropine sulfate.

In the management of mild paralytic ileus Alvarcz and Hosoi have demonstrated that feeding the patient solid food is the greatest stimulus to peristaltic activity.

Mayo and Bartlett advocate the use of spinal anesthesia as a diagnostic procedure to differentiate between mechanical and paralytic ileus. Ochsner calls attention to the fact that this is not a reliable diagnostic aid because an evacuation of the intestines was produced in 16 per cent of the cases with mechanical obstruction and as a result one would be apt to overlook those cases of mechanical obstruction which did respond to splanchnic block. Ochsner demonstrated experimentally that splanchnic anesthesia performed by introducing the anesthetic solution posteriorly in the retroperitoneal space according to the technique of Kappas was effective in overcoming a paralytic ileus following a mechanical obstruction. In every case the splanchnic anesthesia produced an increase in intestinal tonus. Ochsner prefers splanchnic anesthesia according to the technique of Kappas to spinal anesthesia because the effect on blood pressure was less marked and the effect upon intestinal motility was more prolonged. If splanchnic anesthesia is used as a treatment for paralytic ileus Ochsner believes that it is important to omit the preliminary use of blood pressure raising drugs such as epinephrine and adrenalin since these drugs stimulate the myoeuteral junction of the sympathetic system distal to the point of block and thus the desired result might be nullified.

THE EFFECT OF DRUGS ON MECHANICAL INTESTINAL OBSTRUCTION

The only indication for the use of drugs in mechanical intestinal obstruction is to increase intestinal peristaltic activity after the release of the mechanical obstruction. Following the release of the mechanical obstruction, an atonic bowel may advantageously be stimulated to activity by the

use of drugs. Under no circumstances should drugs be used in the treatment of mechanical intestinal obstruction prior to surgical intervention. To do so would result in increasing the peristaltic activity in a bowel which is already hyperactive. The treatment for all cases of mechanical intestinal obstruction is surgery as soon as the patient has been properly prepared.

DISORDERS SIMULATING GASTRO- INTESTINAL OBSTRUCTION

The organic and functional disorders simulating gastro intestinal obstruction are legion. Since the differential diagnosis can sometimes be extremely difficult it is essential that the surgeon be familiar with these various disorders. When one considers that these conditions simulating intestinal obstruction do not require surgery but can be adequately handled by conservative measures, the importance of a correct diagnosis becomes obvious. In this chapter we will cover only the more common of these disorders. In that sense our discussion will by no means be a complete one. Isolated case reports of other disorders which produce the signs and symptoms of intestinal obstruction will undoubtedly appear from time to time.

RUPTURED EPIGASTRIC ARTERY

Spontaneous rupture of the superior or inferior epigastric artery can mimic acute intestinal obstruction. The hematoma formed as the result of a ruptured epigastric artery usually lies in the posterior part of the rectus sheath between the preperitoneal fat and the posterior surface of the rectus muscle and spreads beneath the muscle. Laterally the hematoma is limited by the fascia of the linea semilunaris and by the fascia of the transversalis at this level but below the linea semicircularis the hematoma may spread laterally. It may on occasion bulge into the peritoneal cavity or may push down into the pelvis.

Predisposing Factors

Among the many predisposing factors impli-

cated in the production of this condition the following have been noted:

- 1 Hypertension
- 2 Arteriosclerosis
- 3 Blood dyscrasias such as scurvy hemophilia or leukemia
- 4 Weak abdominal musculature

The actual rupture of the epigastric artery is the result of some trivial strain such as coughing, sneezing or straining during bowel movement. Pregnancy and trauma have been reported as precipitating factors in the production of such hemorrhage when they are associated with one or more of the predisposing factors. In an occasional case no definite predisposing or precipitating cause can be found. This is apt to occur in elderly individuals with atheromatous blood vessels and muscular weakness.

Signs and Symptoms

The symptoms in such cases develop suddenly. There is a sudden severe pain over the site of the hematoma accompanied by nausea and sometimes vomiting. Examination reveals abdominal distention and some rigidity and tenderness limited to the region of the hematoma. This clinical picture of pain, nausea, vomiting, abdominal rigidity, tenderness, and abdominal distention is caused by the stimulation of the parietal peritoneum. This symptom complex is common to hematomas of the rectus sheath as well as to intra abdominal and retroperitoneal hemorrhage. If the hematoma lies below the linea semicircularis it may cause a bulge

ing of the peritoneum which may be palpable transvaginally so that an erroneous diagnosis of an abscess or an ovarian cyst might be made. Numerous cases of this type have been operated upon because of an incorrect diagnosis.

Diagnosis

The diagnosis is based upon the sudden onset of pain associated with the finding of localized tenderness over an ill defined mass. The temperature pulse and respiration are usually normal. The patient looks well but the abdominal findings are suggestive of some acute abdominal catastrophe. There is a normal blood count early in the course of the disease. This may change as the disease progresses so that a leukocytosis due to concealed bleeding, or an anemia due to blood loss may be found in well developed cases.

The diseases most commonly confused with rupture of the epigastric arteries are acute appendicitis, diverticulitis, mesenteric thrombosis, intussusception, volvulus, and acute cholecystitis.

Kinder and MacLennan reported cases in which a rupture of the inferior epigastric artery was diagnosed as intestinal obstruction. These cases were characterized by their occurrence in middle aged women with poor musculature who were so obese that it was difficult to distinguish between a tumor mass in the abdominal wall and a mass within the peritoneal cavity. The presence of such a mass coupled with abdominal distention or the sudden onset of very severe symptoms were the major factors contributing to the erroneous diagnosis of acute intestinal obstruction in such cases.

Treatment

In the management of these lesions involving the inferior or superior epigastric artery, once the correct diagnosis has been made, a conservative policy may be instituted. Ice packs should be applied to the region of the hematoma formation. A compression dressing is often helpful when directly applied over the hematoma. In those cases in which the hematoma is extensive or in which there is evidence of a continuing increase in size it may be necessary to open the rectus sheath, expose the offending epigastric artery, and ligate it. In most cases, however, surgical ligation of a

bleeding vessel is not required and in the main the surgical approach should be reserved for those cases where the diagnosis is doubtful. In the vast majority of cases, a conservative method of treatment without surgery is most desirable. Compression of the abdominal wall at the site of the hematoma is generally sufficient to produce a cessation of bleeding. Awareness of the possibility that a rupture of the epigastric artery may be present is the greatest aid in arriving at a correct diagnosis since most diagnostic errors have occurred because of a failure to consider this condition as a possible cause for the symptoms.

RETROPERITONEAL BLEEDING

Retroperitoneal bleeding regardless of cause may produce a symptom complex simulating intestinal obstruction. It is an established fact that a paralytic ileus may occur in association with the development of this condition. The ileus may be of such a degree that the distention becomes marked. The importance of paralytic ileus as a complication of retroperitoneal bleeding in these



FIG. 413. Marked intestinal distention as a result of retroperitoneal bleeding from an aortic aneurysm.

patients has been repeatedly emphasized by numerous workers. Guibrl and Cuenot proved that blood in the retroperitoneal space acts as an irritant. Although it was first believed that this irritation was sympathetic in origin, the consensus at present appears to be that the mechanism of ileus due to retroperitoneal hemorrhage involves the parasympathetic as well as the sympathetic system. The ileus usually is severe and does not respond to ordinary measures until the cause of the retroperitoneal bleeding is removed.

Causes

There are numerous causes for retroperitoneal hemorrhage. Among these three large groups of cases may be classified:

Traumatic and Surgical Causes. Trauma may be responsible for a retroperitoneal hemorrhage of such magnitude that it results in a paralytic ileus. In this group one may find fractures of the ribs, vertebrae or pelvis.

Retroperitoneal hemorrhage may be secondary to any operative procedure related to the retroperitoneal tissues. Abdominal distention develops soon after. This type of retroperitoneal hemorrhage is of special importance. In this event the diagnosis should be apparent to the operating surgeon. Among the unusual varieties of retroperitoneal hemorrhage from this source are those cases which are the result of appendectomy in which there may be a diffusion of blood retroperitoneally giving rise to a paralytic ileus. In a report by Karabin, the source of the retroperitoneal hemorrhage following an appendectomy was found to be the lateral ascending branch of the deep circumflex artery. This vessel is situated about an inch from the anterior superior spine of the ileum and ascends between the internal oblique and transversus muscles to about the level of the umbilicus. Injury to this vessel may produce a very extensive retroperitoneal hemorrhage. This accident if not recognized may result in an erroneous diagnosis of paralytic ileus resulting from peritonitis as the cause of the intestinal distention.

Retroperitoneal hemorrhage may also occur after hysterectomy. In cases of this type the hemorrhage may develop in the broad ligaments and direct its way around retroperitoneally, pro-

ducing the clinical appearance of intestinal obstruction.

Diseases of Blood Vessels or Blood Dyscrasias Causing Retroperitoneal Hemorrhage. Bleeding from any of the retroperitoneal blood vessels may give rise to this symptom complex. The aorta not uncommonly is responsible for such bleeding, although any retroperitoneal vessel may rupture in an arteriosclerotic or hypertensive individual.

In addition to being a common source of such bleeding, diseases of the aorta may produce severe intestinal distention without retroperitoneal bleeding. That dissecting aneurysms with retroperitoneal hemorrhage may produce paralytic ileus as a result of autonomic stimulation is well known. However, it has not been generally realized that dissecting aneurysms can produce clinical signs and symptoms suggesting a diagnosis of obstruction of the gastrointestinal tract. Warren and McQuown reported an extraperitoneal rupture of the aorta with dissection into the mesenteric arteries which simulated intrinsic disease of the gastrointestinal tract producing the symptoms of mesenteric thrombosis. In a case of this type true intestinal obstruction may result from the intestinal ischemia secondary to the dissecting aneurysm. Shelley reported a case in which the development of an aneurysm in the descending aorta was associated with progressive distention and obstipation. Van Meurs reported a case actually caused by a dissecting aneurysm of the abdominal aorta which was diagnosed as obstruction of the colon because of the severe abdominal pain associated with distention which was not relieved by enemas. Cuny reported a case diagnosed as volvulus of the pelvic colon and operated upon for this disorder, whereas at operation the cause of the abdominal pain and distention was found to be a ruptured aortic aneurysm. Mayo reported a rather unusual case in which the development of marked intestinal distention down to the middle of the sigmoid colon suggested obstruction at this point. There was no gas visible radiologically in the rectum but several gas-filled loops of small bowel were noted. The cause of these findings was a dissecting aneurysm of the abdominal aorta which involved the point of origin of the inferior mesenteric artery. In this

case the signs and symptoms were those of obstruction of the colon. Death was caused by rupture of the aortic aneurysm. Although dissecting aneurysm *per se* without retroperitoneal bleeding can simulate intestinal obstruction, those cases in which such aneurysms are accompanied by bleeding are characterized by an enormous intestinal distention. The abdomen may be markedly distended and tympanic. Auscultation discloses little or no peristaltic activity.

Diseases of the hematopoietic system such as leukemia, hemophilia, and purpura are capable of producing retroperitoneal bleeding which simulates intestinal obstruction.

Diseases of Retroperitoneal Tissue. Any infectious process in the retroperitoneal area, such as perinephritic abscess or perinephritis, neoplasms, and embolic or thrombotic processes may result in the development of a paralytic ileus. Infection in the perinephritic area is a common cause for such ileus. Retroperitoneal tumors, especially those producing irritation of the sympathetic chain, may cause ileus. Emboli or thrombi to vessels in this area are uncommon causes of ileus.

Signs and Symptoms

The onset of retroperitoneal bleeding usually occurs with a sudden pain without any history of previous attacks. The pain may be generalized over the entire abdomen or localized to one side or the other. Not infrequently the pain may radiate to the legs or the back. Following the onset of pain the patient complains of feeling faint and may collapse. At this time there is generally a marked pallor and the blood pressure is low. This may be associated with a rapid pulse. A mass may or may not be palpable transabdominally or a bulging may be noted in the flanks. The abdomen is often rigid and tender at the onset of symptoms, but as the disease progresses the rigidity usually diminishes and an ileus of varying degree develops. Abdominal distention may appear from 24 hours to 5 days after the onset of the retroperitoneal bleeding. This distention may reach enormous proportions, especially in the case of a rupture of an aortic aneurysm. In the case of an abdominal aortic aneurysm of the saccular type a bruit may or may not be heard by abdominal

auscultation. It is rarely heard over the hematoma resulting from a rupture of an aneurysm of this type. Vomiting may be severe.

The laboratory is of little help early in the course of the disease. As the disease progresses, however, anemia may become an important diagnostic point. A survey film of the abdomen may or may not show an abdominal aortic aneurysm, particularly if the aneurysm is of the saccular type. If the aneurysm is dissecting, the survey film will invariably be negative. Kampmeier found that the radiologic examination was diagnostic in 75 per cent of the cases of saccular abdominal aortic aneurysm and that in 70 per cent of the cases there was a definite pressure erosion of the bodies of the vertebra without destruction of the intervertebral discs. This is pathognomonic of an abdominal aneurysm.

Diagnosis

A specific diagnosis may be difficult in these cases, but whenever a diagnosis of paralytic ileus is made in a patient above the age of 60 who presents evidence of severe abdominal pain and shock, a possible diagnosis of dissecting aneurysm should be considered. Hematologic study will be helpful not only in disclosing anemia suggesting such retroperitoneal bleeding but also in disclosing a hematopoietic cause for such bleeding. If radiologic study of the abdomen reveals evidence of an abdominal aortic aneurysm, the diagnosis may be considered as probable. A history of severe trauma or radiologic evidence of fracture of ribs, pelvis or vertebrae is a clue as to the cause of the ileus.

Mesenteric thrombosis, volvulus, and intussusception must be considered in the differential diagnosis. Even though an exact diagnosis of retroperitoneal bleeding as a source of the ileus may be difficult, it is generally not too much of a problem to rule out the organic obstructions referred to. The clinical history, physical findings, radiologic findings, and course of the disease will almost always permit one to rule out mechanical obstruction.

Treatment

In those cases in which retroperitoneal hemorrhage is caused by a bleeding vessel after surgery, an effort should be made to find the vessel and

ligate it. When this is not possible, the retroperitoneal space may be packed.

It is important to recognize that the intestinal distention is due to retroperitoneal bleeding since surgical intervention should be avoided in such cases except in the group referred to above. The intestinal distention must be treated by intubation and operation should not be resorted to even as an emergency procedure. The recent developments in vascular surgery permitting aortic transplants may enable some of these patients to be salvaged. In the past the prognosis in all cases of bleeding aortic aneurysms has been poor.

DISORDERS OF THE URINARY SYSTEM

Tixer and Clavel studied the relationship of the retroperitoneal syndrome to the gastro intestinal reflex on the kidneys. A reflex which they call the renodigestive was demonstrated and a syndrome simulating peritonitis was developed experimentally by stimulation of the kidney and the retroperitoneal tissues. Although the exact mechanism of this reflex could not be explained, it was suggested that it involved several factors. It was either a summation of the irritating impulses, an individual predisposition or a sensitization of nerve centers through an infection or intoxication.

Ureteral colic caused by a stone in the ureter is not infrequently associated with the development of paralytic ileus. Trauma to the ureter due to cystoscopic examination may at times cause a severe degree of paralytic ileus. Uremia is not uncommonly associated with paralytic ileus and marked intestinal distention. Pyelitis and pyelonephritis sometimes produce signs and symptoms suggestive of acute intestinal obstruction. In an occasional case seminal vesiculitis, particularly in patients who have been catheterized repeatedly, may result in marked intestinal distention associated with abdominal pain. In such cases the tenderness over the seminal vesicles found on rectal examination should suggest the possible diagnosis. The presence of a high fever and leukocytosis early in the course of the disease make the diagnosis probable. However, difficulties can arise when acute seminal vesiculitis is found in an individual presenting a large hernia. In cases of this type it may be difficult to decide whether the signs



FIG 414 Severe paralytic ileus following biliary tract surgery as a result of a cystoscopic examination on the eighth postoperative day.

and symptoms of bowel obstruction are produced by the hernia or are the result of the vesiculitis. In any doubtful case it is better to subject the patient to surgical intervention so that an accurate diagnosis can be made.

CORONARY ARTERY AND HEART DISEASE

At times coronary occlusion may manifest itself in such fashion that it simulates acute intestinal obstruction. The intestinal distention may be very marked and the patient may complain of severe abdominal pain. In many of these patients the intestinal distention can be marked and abdominal tenderness sometimes appears so that a strangulating obstruction is simulated. A careful examination of the heart, an electrocardiographic examination, a review of the survey film of the abdomen and the clinical picture should lead one to a correct diagnosis.

When confronted with a patient presenting known coronary disease who complains of abdominal pain tenderness and distention it may be difficult to decide whether all the abdominal findings are due to the known coronary disease or whether the patient has mesenteric occlusive vascular disease in addition to his coronary disease. There are no hard and fast rules for making a diagnosis in all such cases. The surgical judgment and the experience of the examining physician alone can determine whether the patient is mechanically obstructed.

Although it has been repeatedly emphasized that in the management of acute small bowel obstruction surgery should be performed at all times when intestinal obstruction is suspected this rule does not apply to those cases where there is an extensive myocardial involvement or where a question of coronary disease is present. In cases of this type the risk of surgery may outweigh the risk of the obstruction. A conservative policy should be followed for at least the first 24 hours. In the meantime correlation of all the evidence and repeated examinations of the patient will generally yield a correct diagnosis.

The laboratory is of little help in the differential diagnosis. A leukocytosis can occur with either coronary occlusion or occlusive vascular disease. In addition an electrocardiographic study may be negative when the patient is admitted to the hospital but 12 to 24 hours later may show definite changes indicating the true diagnosis.

Myocardial failure not uncommonly causes intestinal distention by producing edema of the bowel wall. An atony of the colon may be produced as a result of myocardial failure particularly in elderly individuals and may result in the accumulation of large amounts of fecal material in the colon. Attempts to clean out the lower colon by means of high colonic irrigations may have to be deferred because of the poor condition of the heart. The probable cause of the intestinal atony is the tissue anoxia produced by the decreased oxygenation associated with the myocardial failure. A knowledge that intestinal distention or many of the signs of intestinal obstruction can occur with myocardial failure is of importance in that

the attending physician will not be misled into an erroneous diagnosis of intestinal obstruction.

The treatment of both these cardiac causes of intestinal distention consists of simply treating the cardiac pathology. In the majority of cases once the patient is over the myocardial failure the colonic atony greatly improves.

LESIONS OF THE SPINAL CORD

Lesions of the spinal cord may produce atonic paralysis of the intestine by cutting off its central nervous motor supply. The gastro-intestinal tract may become completely paralyzed following a fracture of the spine where the cord is compressed high up in the dorsal region. The atonic paralysis that develops in the gastro intestinal tract results in a tremendous intestinal distention. This may be permanent if the cord has been completely crushed or may be followed by more or less of a recovery of tonus if the cord lesion is only partial. A fracture of the tenth or twelfth dorsal vertebra with forward flexion and angulation of the spine may produce temporary paralysis of everything below the twelfth thoracic vertebra. In such cases the abdominal distention usually clears up after the fracture has been reduced. The fact that the abdominal distention usually clears up within two or three days after immobilization indicates that the compression of the cord due to angulation has occurred with only temporary suspension of the transmission of nervous impulses.

Mediastinal lesions involving the vagus and sympathetic afferent nerves to the intestine may cause paralytic ileus.

FRACTURED RIBS

Fracture of the ribs may cause the development of paralytic ileus. Adams reported the first two cases of this type in 1910. Since then isolated case reports have appeared from time to time. The exact mechanism by which paralytic ileus was produced as a result of a fracture of one or more ribs is not clearly understood. Adams believed that some displacement of the proximal portion of the fractured ribs occurred producing irritation of the sympathetic chain of the thoracic ganglia and the splanchnic nerves arising from them. There is

some difference of opinion with regard to this point Vandel pointed out that anatomically it is difficult to understand how the proximal rib fragment which is strongly bound to the vertebral column by ligaments could move to the extent required for the explanation of irritation to apply On the basis of their studies Altemeier and Wadsworth believed that there were four possible means of splanchnic stimulation

- 1 Irritation of the sympathetic thoracic ganglia and therefore the splanchnic nerve may occur as a result of displacement of the proximal rib fracture segment (Adams theory)
- 2 Edema of the tissue adjacent to the point of fracture may encroach upon the position of the thoracic sympathetic ganglia
- 3 Retropleural hemorrhage in the vicinity of the thoracic sympathetic ganglia or splanchnic nerves may result from a trauma and may produce pressure on the sympathetic ganglia and the splanchnic nerves
- 4 Stimulation of the peripheral nerves either intercostal or abdominal may result in a reflex stimulation of the splanchnics through the thoracic ganglia

ACUTE PANCREATITIS AND CHOLECYSTITIS

Acute pancreatitis may produce a marked distention of the transverse colon simulating obstruction of the splenic flexure This has been observed by Hulton who mentions it as a diagnostic point This phenomenon is not constant in acute pancreatitis The intestinal gas pattern of paralytic ileus associated with acute pancreatitis is variable It varies from the distention of one or more loops of small bowel to a marked distention of the entire gastro intestinal tract Early in the course of the disease an accurate diagnosis may be difficult although a marked elevation in the serum amylase is helpful in arriving at a correct diagnosis

Acute cholecystitis can simulate acute intestinal obstruction A survey film of the abdomen may show one or more loops of distended small bowel or at times the intestinal distention may be extensive This is particularly true of those cases in which phlegmonous cholecystitis or walled off ab-

cess around the gall bladder is present A carefully taken history will usually permit one to arrive at a correct diagnosis

The ileus following acute pancreatitis and cholecystitis must be treated by conservative measures using the long intestinal decompression tube The importance of arriving at a correct diagnosis differentiating these entities from intestinal obstruction lies in the fact that acute pancreatitis is not a surgical lesion and should be treated by conservative means The same is true for acute cholecystitis unless it is seen early in the course of the disease or unless signs of impending perforation appear

DIAPHRAGMATIC PLEURISY AND PNEUMONIA

Diaphragmatic pleurisy may be mistaken for intestinal obstruction at times The diaphragmatic pleurisy may cause a paralytic ileus which may simulate bowel obstruction The pain and tenderness in the side of the abdomen caused by the pleurisy when associated with marked intestinal distention further suggest bowel obstruction Because the abdominal musculature on the side of the pleurisy goes into spasm in an effort to limit the excursion of the diaphragm a diagnosis of strangulating obstruction is not uncommonly made The presence of fever pleural friction rub, leukocytosis, and the radiologic chest findings make a correct diagnosis possible An additional diagnostic point is the fact that pleurisy is always associated with an elevation in temperature whereas in the acute stage of mechanical intestinal obstruction there is never any primary elevation of temperature

Similarly pneumonia is a not infrequent cause of paralytic ileus particularly in infancy and childhood and may at times be mistaken for acute intestinal obstruction

NUTRITIONAL ELECTROLYTE AND VITAMIN DEFICIENCIES

Nutritional Deficiencies

Nutritional deficiencies may at times produce gastro intestinal changes simulating intestinal obstruction Jones and Eaton noted the effect of edema on gastro intestinal motility in cases sub-

jected to surgery presenting a low plasma protein concentration. In these cases because of the preoperative starvation as well as the excessive intravenous saline after surgery changes in the intestinal motility were demonstrated. Gastrointestinal stasis and a resultant intestinal distention occurred. Kaydin in studying this subject demonstrated that the gastric emptying time was prolonged in experimental animals as well as humans when the serum protein content was lowered. In addition Kaydin and Frank demonstrated that the small bowel emptying time of experimental animals was seen to increase as the serum protein concentration was reduced by repeated bleeding. In the patients in whom surgery was associated with edema of the bowel it was found that there was a definite delay in intestinal motility.

Patients suffering from liver disease nutritional deficiencies uremia due to renal failure and extensive burns often develop a definite delay in intestinal motility. This leads to the development of distention. In this type of case the impairment of small bowel motility is associated with edema of the bowel. Patients who have a partial intestinal obstruction may become completely obstructed as a result of such intestinal edema.

Vitamin Deficiencies

Thiamin chloride deficiencies may cause severe and uncontrollable abdominal distention which may lead to an erroneous diagnosis of acute intestinal obstruction and result in needless surgery. Disturbances of the digestive tract associated with vitamin B deficiencies cause a variety of symptoms so that the clinical picture is not constant. Brown and Trowell have reported a syndrome seen in Polish refugees and in African and Indian adults and children in which the radiologic picture was similar to that described by Golden. The symptoms in these cases were suggestive of pyloric obstruction. In addition great gaseous distention of the colon was common. The digestive symptoms accompanying vitamin B deficiency may manifest themselves in various syndromes.

Leithauser described atypical ileus as a result of a nutritional deficiency of thiamin hydrochloride. He believed that this was more likely to occur in individuals suffering from liver disease. In the

cases studied by Leithauser tremendous intestinal distention was the chief finding. The distention was not controlled by mechanical decompression or by the administration of Prostigmin but it did respond dramatically to the administration of thiamin hydrochloride and vitamin B complex.

Electrolyte Deficiencies

A low blood potassium produces the effect of paralytic ileus even in the presence of a normal blood protein. A loss of potassium often occurs following surgery and may be aggravated by excessive administration of intravenous saline. The first signs of hypokalemia appear when the potassium level descends to 3.5 mEq per liter and at 2.6 mEq or less the symptoms become severe. The intestinal distention in these cases is not relieved by Prostigmin which suggests that the amount of acetylcholine available in the bowel wall is not sufficient to affect the muscularis. The proper concentration of potassium ions is necessary for the production of acetylcholine and therefore for the transmission of nerve impulses. Experimental evidence suggests that potassium accelerates the production or the release of acetylcholine which is counteracted by calcium and magnesium. In addition movement of potassium ions plays a part in the transmission of nerve impulses. Intestinal distention with a radiologic picture of paralytic ileus may be due to changes or disease in the bowel wall or its mesentery as well as to the lack of potassium.

Treatment

The treatment of the ileus that results from disorders due to the members of these deficiency states consists of correcting the deficiency state. Intestinal intubation with a long tube will keep the gaseous distention down until the primary deficiency state is corrected.

As long as a plasma protein concentration remains at the edema level therapy directed toward fluid and salt replacement will tend to increase the edema. On the other hand therapy directed toward raising the plasma protein concentration will restore the normal bowel activity and aid in deflation of the patient.

Nutritional deficiency should be suspected and a

therapeutic trial of thiamin and vitamin B complex made in cases of abdominal distention in which the evidence does not justify a positive diagnosis of mechanical obstruction of the bowel. It is probably wise to administer thiamin hydrochloride to those patients whose nutritional status is at all questionable or in whom there is any suspicion of liver damage.

Potassium deficiency can be readily corrected by the administration of proper amounts of this element. Once the diagnosis has been made by proper laboratory study the treatment becomes a simple matter.

FUNCTIONAL DISORDERS OF THE SMALL INTESTINE AND COLON

Many surgeons have pointed out that functional disturbances may cause abdominal distention simulating intestinal obstruction. Such cases are found more frequently in women although an occasional case may be found in a man. The majority of these cases have been reported in virgins or women who have not borne children. In these cases the intestinal distention usually began immediately or soon after abdominal surgical maneuvers and generally temporary relief followed further surgical manipulation. The impression gained by most authors is that the basic dysfunction in these cases is one of the motor mechanism of the large bowel.

Hysterical Distention

Hysterical abdominal distention may simulate acute intestinal obstruction. This type of abdominal distention has been known for over 100 years and the pseudopregnancies are a manifestation of this entity. In these cases the abdominal distention and the symptoms are so closely suggestive of obstruction of the bowel that unnecessary surgical intervention may be resorted to.

Diagnosis. There are certain clues that suggest hysterical distention rather than organic disease and thus lead one to a correct diagnosis.

- 1 There is a history of vomiting but no dehydration or evidence of fluid loss can be found on physical examination.
- 2 The patient complains of severe pain in the abdomen but the temperature, pulse and leukocyte counts are normal.

- 3 Normal tympanitic quality of the abdomen is found on examination. An occasional case may have gaseous distention of the bowel.
- 4 Distention which may be found on physical examination is out of proportion to the amount of gas observed on the abdominal X-ray survey film.
- 5 Pronounced lordosis is found. This is caused by thrusting the abdomen forward.
- 6 A complete remission of the abdominal distention occurs when the patient is under general or spinal anesthesia.

Differential Diagnosis. Great care must be taken to differentiate this hysterical type of abdominal distention from true mechanical intestinal obstruction. In any patient in whom hysterical intestinal distention is suspected there may also be an associated mechanical obstruction. Alvarez has pointed out that the pronounced distention associated with hysterical patients is not due to any excessive gas in the digestive tract but is due to a contraction of the muscles of the back and the upper part of the abdominal cavity with a relaxation of the muscles of the anterior abdominal wall. As a result of this the changes associated with assuming a lordotic posture tend to throw the abdominal contents forward and somewhat downward toward the pelvis.

It has long been known that this distention disappears rapidly and suddenly without the passage of gas when these patients are given a general anesthetic. In those cases in which hysterical abdominal distention is associated with air swallowing the patient may pass large amounts of gas through the gastro intestinal tract. Although in most cases in which the symptoms resemble those of true intestinal obstruction a differentiation can readily be made in an occasional case differentiation may be so difficult that the surgeon is forced to explore the abdomen in spite of a suspicion that the patient may be suffering from hysteria.

Although these patients present what seems to be a history of intestinal obstruction (for example vomiting of several days duration) they appear relatively well clinically. They are not dehydrated, their tongues are moist, the skin is of normal texture and they do not appear to be acutely ill. They may complain of severe abdominal pain and

tenderness so that one may suspect a strangulating type of obstruction. Yet laboratory studies show a normal temperature, pulse and no changes in the blood picture. In addition, the distended abdomen may not be unusually tympanitic and the radiologic survey film fails to show small bowel gaseous distention.

Treatment. These patients are best treated surgically for permanent results. The abdominal findings can almost invariably be cleared up by the use of a general anesthetic, spinal anesthetic or bilateral splanchnic block.

Functional Intussusception

Functional intussusception may occur without organic disease of the bowel wall. Teitelbaum and Aronson have demonstrated that recurrent self-reducing intussusception in the small bowel of children can be demonstrated radiologically. Golden has noted the same manifestations in adults and also in a number of young children with celiac disease. These are believed to be examples of functional intussusception in a psychosomatic type of individual. Golden reported a case of distention suggesting a paralytic ileus which appeared eight days following amputation of a carcinomaous breast in a 75-year-old woman. No organic cause for the persistent distention could be found. The administration of Urecholine was promptly followed by the disappearance of distention with an uneventful recovery.

UNUSUAL MISCELLANEOUS DISORDERS

This group which may cause paralytic ileus consists of a miscellaneous assortment of disease entities. However they all have one thing in common—their ability to produce an intestinal distention simulating intestinal obstruction. In *modus operandi* and treatment each member of this group differs widely from the next.

Amyloidosis

Amyloidosis of the intestine can present a picture of ileus and may result in an operation for intestinal obstruction. Golden reported several cases of this type. In the cases reported amyloid deposits were found in the intramural nerve ganglia of the small intestine and in the muscle

fibers of the intestinal wall. The radiologic picture was that found in intestinal obstruction. Persistent gaseous distention and a slow transit time of the barium through the intestinal tract were reported. Patients with this disorder show little or no response to Prostigmin or to Urecholine. A lack of response to Prostigmin or Urecholine should suggest that the muscle is unable to react because of disease of the intestinal wall.

Organic Disease of Mesentery

Organic disease of the small bowel mesentery may cause intestinal distention. Tremendous dilatation of the ileum may at times be found associated with and apparently caused by malignant infiltration of the mesentery. The explanation offered in such cases is the denervation of the small bowel by the malignant infiltration of its mesentery.

Persistent Spasm of Colon

A persistent spasm of either the transverse or descending colon is not infrequently diagnosed as acute intestinal obstruction. The diagnosis in this type of case may be made on the basis of the patient's history and the fact that he is visibly a nervous and apprehensive individual. There is usually a history of epigastric distress or of alternating constipation and diarrhea of many years duration. These patients respond to a bland nonirritating diet and to the use of tincture of bella donna or atropine and phenobarbital.

Diabetic Acidosis

Diabetic acidosis may at times be associated with nausea, vomiting and abdominal distention. Unless this is kept in mind a diagnosis of intestinal obstruction could readily be made and unnecessary surgery performed. The ileus associated with diabetic acidosis can be clinically recognized by the fact that although the radiologic evidence shows marked small bowel distention, intestinal peristaltic sounds appear to be normal and the patient's bowels move normally. In addition the finding of ketone bodies in the urine and a high blood sugar is sufficient to make a correct diagnosis. A correction of the diabetic acidosis results in a prompt subsidence of the intestinal distention.



FIG. 415 Note marked intestinal distention in a patient with severe diabetic acidosis. The distention responded promptly to adequate treatment of the diabetic acidosis.

Poliomyelitis

Poliomyelitis during its acute stages may present a picture of intestinal obstruction due to the marked paralytic ileus. This may involve the stomach to a marked degree or may involve the

entire gastro intestinal tract. In such cases a diagnosis of volvulus of the cecum has sometimes been made because the tremendous distention of the stomach has been mistaken for a misplaced cecum. It must be borne in mind that poliomyelitis can produce a paralytic ileus which should be treated conservatively with a long intestinal decompression tube.

Lead Poisoning

Lead poisoning may be associated with uncoordinated muscular spasms of the intestinal tract giving rise to a severe colic. This may mimic intestinal obstruction in its early stage. In addition lead poisoning may cause nausea, vomiting and obstipation. Normally lead colic can be distinguished from intestinal obstruction because distention is seldom a prominent feature in cases of pure lead colic. However, varying grades of colonic and small bowel distention, discernible both on physical examination and radiologic survey, have been described in a few cases of uncomplicated lead colic. In cases such as these it may be extremely difficult to differentiate between a case of pure lead poisoning and intestinal obstruction. The therapeutic test using intravenous calcium is of great help in the differential diagnosis. If the case is one of pure lead poisoning the symptoms of colic clear up almost immediately. If the symptoms are due to a superimposed organic intestinal obstruction, however, the colicky pain, nausea and vomiting continue.

NURSING TECHNIC IN THE MANAGEMENT OF THE OBSTRUCTED PATIENT

One of the most important facets in the treatment of the obstructed patient is the quality of nursing care he receives. Although the nursing care may be routine for a patient suffering from nonstrangulating obstruction seen early in the course of disease and operated upon promptly the obstructed patient seen relatively late in the course of the disease or the patient with an intestinal obstruction that requires a colostomy demands special nursing care. There are a number of special duties accompanying the management of the obstructed patient which are not required for other types of surgery. Patients with intestinal obstruction who show a marked degree of intestinal distention or those in whom preliminary colostomy is required present to the nursing staff three distinct problems in nursing, each of which must have adequate attention if the best results are to be obtained. These problems are: (1) the care of the patient; (2) the care of the decompression tube or colostomy; and (3) the care of the suction equipment used to decompress the patient.

THE CARE OF THE INTUBATED PATIENT

In general patients admitted to the hospital with intestinal obstruction are very sick people. The degree of sickness varies from an acute process in which the patient is writhing with pain to the advanced type of strangulating obstruction in which the patient presents some degree of shock. All these patients are apprehensive and they are all aware of the fact that they are seriously ill. In the management of this type of case a tactful approach by the nursing staff coupled with an

effort to make the patient feel that we are keenly interested in his welfare is of the greatest importance. These acutely ill patients must never be made to feel that they are just another case. This is particularly important in the management of intestinal obstruction in the aged. These individuals become discouraged very easily and have a tendency to become apathetic. The ability to impart to the patient a feeling that he is wanted and that the entire staff is making every effort to make him well again will do much to put him in the proper frame of mind to endure the vicissitudes that lie before him.

Nursing Care Before Intubation

It is essential that the entire nursing staff realize the seriousness of the disorder so that medications are given promptly and every effort is made to keep the patient comfortable. In the elderly patient constant vigilance must be exercised to see that the patient does not develop pressure sores or decubitus ulcers. In the pre-operative period a cheerful attitude should be maintained so that the patient will not fear the impending surgery. Patients admitted with intestinal obstruction may be vomiting. The nursing care in this situation does not consist simply of placing a kidney basin on the patient's bedstand and leaving him to his own devices. If it is at all possible a nurse should be almost constantly in attendance or wherever this is not feasible at least within calling distance at all times. It is highly desirable to assist the patient in every way during the vomiting spells to prevent the possibility of aspiration of the vomitus.

into the trachea with the resultant development of atelectasis. For this reason both the head and the patient's head should be held by the nurse. The linen may require frequent changing during this period of time in order to keep the patient and his bed free of vomitus.

In addition to the small niceties required in the treatment of the sick patient the patient with intestinal obstruction presents a second problem—the stoppage of the gastro intestinal tract producing a severe colicky pain. Sedation of the patient with the subsequent relief of pain is one of the important tasks of the nursing staff. Great care must be exercised so that no sedation is given until a specific diagnosis has been made or unless specific orders have been left by the attending surgeon. The indiscriminate use of morphine during the diagnostic stage of this disease will do much to mask the clinical symptoms so that a diagnosis may not be made until relatively late. An accurate intake and output record must be kept. This is particularly important in the obstructed patient who is losing large amounts of fluid and electrolytes by vomiting as well as within the lumen of the bowel. In those patients in whom a long intestinal decompression tube is passed an accurate intake and output record is of additional importance. The daily output of urine should be carefully scrutinized. A urinary output of 800 to 1000 cc of fairly normal concentration 1020 or less is a good assurance that there is adequate hydration. With the urinary output in this range there will be no nitrogenous retention because of fluid loss. When the urinary output falls below this level and the concentration of the urine increases it should be brought to the attention of the attending surgeon. Intravenous fluids must immediately be increased in order to adequately hydrate the patient.

The care of the intestinal obstruction *per se* may involve the use of the long intestinal decompression tube. This carries with it specific nursing problems. The nursing care for this type of patient is basically a problem of caring for an individual who in addition to his primary illness is being taxed by the inherent problems of intestinal intubation. The nursing care required varies considerably according to the type of tube used for

intestinal decompression. Small lumen tubes like the Miller Abbott tube require considerable nursing care in order to keep the suctioning lumen patent. Other single lumen tubes like the Cantor tube or Harris tube may require little or no nursing care. The nurse should be familiar with the different types of tubes and must know just how much attention is required with each one.

Up to the time the patient is intubated the nursing care follows the usual routine for the specific illness. In the intubated patient the routine tasks take on lesser importance and at times keeping the intubated patient sedated is highly undesirable. The problems that arise in the management of the patient who is being intubated begin at the moment the tube is brought to the patient's room.

Nursing Care During Intubation

When the intestinal decompression tube is brought to the room of the patient in the absence of the surgeon the nurse in charge should check it to be certain that it is the tube ordered for that specific patient. The tube must then be examined to be certain that it is clean and has been properly sterilized. It is a good idea for the nurse to run an applicator into the lumen of the tube to be certain that none of the sterilizing solution is still present. It might also be advisable in case of doubt to rinse the lumen of the tube with tap water as an added precaution or to send the tube back to central supply. Although it might seem that these precautions are unnecessary, failure to take them may have serious consequences for the patient. In one case in which these precautions were not taken the following data was obtained after the death of the patient:

H. S. primipara age 35 a white female was admitted to the hospital because of pregnancy. The conditions were such that cesarean section was decided upon and this was done. Following surgery there was an unusual degree of distention because of paralytic ileus. A long intestinal decompression tube was passed as a treatment for the paralytic ileus. Within 12 hours following intubation the patient went into shock. In 48 hours the patient had expired. An autopsy of this patient revealed a gangrenous type of gastritis. A fairly large amount of lysol solution was found in the stomach. In reviewing the possible source for the introduction of the lysol into the stomach it was found that in central supply the intestinal tube had been

soaked in a sterilizing lysol solution in which the nurse in charge had failed to flush out. As a result a large amount of lysol solution was introduced with the tube into the stomach of the patient.

Once the tube is examined by the nurse and found to be clean and the tube ordered by the surgeon it is ready to be used. Before inserting it the ends of the tube should be examined especially in the case of the Miller Abbott and other double lumen tubes to make sure that the adaptors are properly placed. It is not unusual to find the adaptors improperly inserted into the end of the tube so that the side of the adaptor indicating suction is actually placed in the inflation lumen and the side of the adaptor indicating inflation is actually placed in the suction lumen. When this happens the injection of air or fluid fails to inflate the balloon and the injection of fluids to irrigate the tube inflates the balloon to such an extent that it may be impossible to remove it.

The tube is now ready for use. It is current practice to place the tube in a dish of cracked ice before using. We discourage this practice in the use of the Cantor tube as we have had much better results without freezing the tube. Such freezing makes the tube stiff and uncomfortable. This is especially true when a plastic tube is being used. Our experience has shown that a tube inserted at room temperature is tolerated better by the patient than one placed in a dish of ice.

After the tube has been passed into the stomach of the patient the nurse in charge must see that the instructions relative to the change in position of the patient are carried out exactly as ordered. There is no room for improvisation. When the instructions read elevate the foot of the bed 12 to 15 inches the nursing staff must see to it that the foot of the entire bed is elevated 12 inches and not just the bedspring which may be cranked up. The willingness of the nursing staff to follow orders to the letter in these maneuvers and to pass only as much tubing as is indicated will invariably result in successful intubations. In those institutions in which an intubation team is present all these procedures are performed by interns and residents. In this event the nurse can devote her entire attention to the patient. In many of the smaller institutions long intestinal decompression

tubes are successfully passed by the nursing staff. This is by no means a difficult procedure and a very high degree of proficiency and hence a high rate of successful intubations are achieved by the nursing staffs assigned to this duty.

Nursing Care with the Tube Down

Once the long intestinal decompression tube is well down the gastro intestinal tract the care of the remaining portion of the tube and the method of feeding it downward are important. It should be remembered that the nasal and the pharyngeal mucosa are unaccustomed to the presence of a rubber tube. The presence of this type of foreign body produces an irritation with an outpouring of mucus. This discharge tends to crust upon the tube so that its further downward movement becomes very painful and difficult as long as the crusts are present. This can be avoided to a great extent by administering 2 per cent ephedrine in oil with 0.5 per cent Pontocaine into the side of the nose in which the tube lies. In addition the portion of the tube just beyond the nose should be moistened for a distance of 3 to 4 inches with mineral oil. By constantly keeping this portion of the tube moist there is much less frictional irritation as the tube is advanced.

The pharyngeal mucosa should be kept moist by a judicious intake of saline solution. Under no circumstances should water be given freely by mouth. The intake of water by mouth washes out the stomach and takes the chloride with it. As a result the degree of chloride depletion becomes greatly exaggerated. With the oral use of normal cool saline much less chloride depletion is produced. The position of the tube in the pharynx should be checked from time to time in an effort to keep it away from the Eustachian ostia. This can be done by turning the head from side to side and anteflexing the neck. A contact of the rubber tube with the ostia of the Eustachian tubes may create sufficient irritative edema to result in occlusion and often otitis media.

The nursing care of patients known to be smokers requires more attention than that of non-smokers. In the former group there is generally some degree of pharyngeal irritation even before the introduction of the tube. These patients in

variably present overactive glands along the tracheobronchial tree. The presence of a foreign body such as a long tube for any length of time further increases the irritation and as a result there is an increase in the glandular secretory activity. The accumulation of this secretion in the nasopharynx and in the bronchial tree may result in bronchial plug formation. This is followed by atelectasis. The greatest stimulus for the removal of the bronchial secretions and mucous plugs is ambulation. Although the same sequence of events may occur in non smokers it occurs to a lesser degree because there is less pharyngeal irritation and bronchial secretion prior to intubation. To avoid this complication of intubation two things are required: (1) adequate hydration of the patient to prevent mucous plugs and (2) motion of the patient either in bed or preferably by ambulation to facilitate expectoration of the tracheobronchial secretions.

With a decompression tube well down the gastro intestinal tract the patient loses large amounts of fluid by suction. In some cases as much as 5000 cc. of liquid with particulate matter may be removed in a 24 hour period. Failure to correct this constant loss of fluid may soon result in an increased dehydration with its resultant dangerous consequences. In charting the output by suction it is important to recognize that the oral fluid intake must be deducted because most of this liquid intake is soon aspirated by the tube. The replacement of the fluid loss by intravenous alimentation is essential. Frequent checks on the blood chlorides are essential in any patient being intubated. After the intestinal decompression tube has passed well down the gastro intestinal tract and the intestinal distention is under control, then oral feeding of concentrated liquid food is permissible. During this time the tube may be clamped for two hours after each feeding. Some of the intake will be absorbed by the patient so that less fluid is required intravenously. Jello, beef broth and liquid foods of any type may be given at the proper time in this way. If clamping of the intestinal tube results in any return of the distention this practice must be stopped at once. The most effective way of correcting protein deficiency is by feeding patients ground meat by mouth. The use of amino

acids intravenously will do little to correct hypoproteinemia.

Many intubated patients are apprehensive partly because of their primary illness and partly because of the presence of the tube. Although some patients accept the situation calmly others become upset emotionally. An occasional patient becomes quite antagonistic and will not permit the tube to remain *in situ*. In this event the tact and the understanding of the surgeon and especially the nurse in charge can do much to make the patient cooperative. Many of these patients when properly handled will permit continued intubation.

In the management of the apprehensive patient a small amount of sedation is permissible but over sedation results in a decrease in motion of the patient and is therefore highly undesirable. Patients being intubated must be encouraged to move freely in bed and whenever possible must be ambulant. An encouraging cheerful attitude on the part of the nursing staff during the early period of intubation and careful attention to detail will result in successful intubation with great improvement in the intestinal distention within a 24 hour period. The feeling of well being which accompanies this improvement in intestinal distention is such that most patients become cooperative. However the occasional case which does not respond to intubation because of a marked aversion to the intestinal decompression tube should be recognized and some other form of intestinal decompression should be instituted.

The irrational patient may be a problem to the nursing staff. Many of these patients during their period of clouded consciousness pull out the tube. Any attempt to intubate a patient in coma or stupor or who is otherwise unable to cooperate is doomed to failure from the onset.

In no case should the tube be fastened to the nose of the patient. To do so is to invite complications. The only exception to this rule is the patient in whom a long intestinal decompression tube has been passed prior to the onset of stupor. In this event the tube may be fastened to the patient's face and restraints should be applied in those instances in which the patient becomes irrational. These patients often thrash around in bed to such an extent that one need not concern himself about

the motion of the patient. With the patient lying quietly in a comatose state adequate nursing care requires frequent changes in the position of the patient.

CARE OF THE DECOMPRESSION TUBE

After Intubation

After intubation the care of the long, intestinal decompression tube varies with the type of tube. The small luminal diameter of the Miller Abbott tube requires irrigation with saline every three to four hours to prevent plugging by the liquid particulate matter found in the gastro intestinal tract. Before doing this one must be certain that the solution is really connected with the decompressing lumen otherwise the irrigating large lumen tube like the Cantor tube does not require such nursing care. The fluids that the patient drinks are readily aspirated by the tube keeping it flushed clean at all times. In the Cantor tube the size of the holes, their elliptical shape and the fact that they are strung along a distance of 2 feet of tubing, render them practically immune to plugging.

Failure of the intestinal tube to suction out intestinal contents requires immediate attention by the nurse in charge. First check the source of negative pressure to be certain that it is working and adequate. If the suction is working, and the tube still does not bring up intestinal contents it should be irrigated with saline in order to determine its patency. If saline cannot be freely injected through the tube then its lumen is plugged. At times withdrawal of several inches of tube corrects the angulation or kinking, thus restoring the obstruction of its lumen. It is not uncommon for many tubes, particularly those which have been used a number of times to be sharply angulated and obstructed especially when too much tubing is passed at any one time. Almost invariably with a drawal of 2 to 4 inches of tube will correct this problem.

After Removal

When the decompression tube has been removed from the patient it is often covered with mucus and intestinal contents. The porous latex bulb on

containing the mercury must be stripped off and discarded. The mercury is washed and returned to its container. The tube is thoroughly flushed and rinsed in water. It is then sent to central supply where it is put through the following routine:

- 1 Suction is applied through the tube with soap water or detergent until clear.
- 2 The tube is then suctioned through and thoroughly rinsed with tap water.
- 3 The tube should be then rinsed thoroughly with distilled water.
- 4 Soak the tube for 24 hours in zephiran aqueous 1:1000.
- 5 Then rinse with tap water.
- 6 Soak the tube for 24 hours in rose water to remove all odors.
- 7 Suction the tube through with distilled water.
- 8 Hang the tube up to dry for 48 hours.
- 9 When the tube is dry reserve it with a new balloon. A new balloon is used for each intubation.

Although this method appears to be time consuming, it not only sterilizes the tube but removes all odor and thoroughly cleanses it.

CARE OF THE SUCTION EQUIPMENT

Constant attention should be given to the source of negative pressure regardless of the type of tube used. This is particularly important in the use of Wangenstein's water displacement method. There is little value in having an intestinal decompression tube far down the gastro intestinal tract when the source of negative pressure at the end of the tube is not working. In a situation of this type the patient is far worse off than if no tube were being used.

The various connections between the decompression tube and the source of negative pressure must be air tight. Leakage in the tubing or at the connections results in a loss of negative pressure and hence a poorly functioning decompression. Constant vigilance must be exercised to keep the source of negative pressure in constant operation from time to time the glass connecting tube between the long intestinal decompression tube and the source of negative pressure should be checked.

to determine whether intestinal contents are being aspirated properly. A lack of drainage when noted in the glass connecting tube should result in a careful check being made of the source of negative pressure to determine whether it is properly functioning and if so attention should be directed to the long intestinal decompression tube to determine whether it is obstructed.

CARE OF THE PATIENT AFTER SURGERY

When the patient returns from surgery a nurse must be in attendance until the patient is well reacted. Vomiting and aspiration of vomitus from the mouth and nose must be watched for and prevented by carefully suctioning out the mouth and nose at frequent intervals with a Yankauer suction tip. A patient may be successfully operated upon for intestinal obstruction only to drown in his vomitus upon his return to the ward. Under no circumstances should a patient operated upon for intestinal obstruction be allowed to remain unattended until he is fully reacted. When he is reacted and if there is no vomiting the patient may be left alone. In most modern hospitals a recovery room is provided so that the nursing personnel there assume the responsibility for this care. The intestinal decompression tube should be immediately connected to the source of negative pressure, a check made that the apparatus is functioning and then the patient may be left safely.

Those obstructed patients who have been successfully operated upon and who are not unduly distended may require only routine care from this point onward until discharge from the hospital. However patients with neglected intestinal ob-

struction who are admitted with considerable distention should be closely supervised throughout their hospital stay. The incision should be checked at least twice a day because of the well known tendency of such distended and poorly nourished patients to eviscerate. Any serosanguinous drainage from the incision should be noted and called to the attention of the surgeon. This is often an indication of early wound separation. After notifying the surgeon immediately the wound dressing should be reinforced and a tight binder applied. In the event that complete separation of the wound has occurred with evisceration a fast call should be placed for the resident and a sterile dressing applied. The patient should receive an injection of some sedative drug immediately in order to assure his cooperation and to keep him quiet.

The patient with a colostomy must receive special attention. The abdomen must be carefully cleaned after each evacuation from the colostomy. The skin around the colostomy should be carefully tended to prevent the development of a weeping, oozing type of dermatitis. In some instances, a colostomy paste may be applied to the skin just around the colostomy opening. If the intestinal contents evacuated from the colostomy are semisolid the skin will require little or no additional care. When the intestinal contents are liquid however considerable nursing care to the skin about the colostomy is required since it is this type of drainage that is so apt to cause severe irritation. The best method of treatment for such skin changes is to prevent their occurrence. This can be done by scrupulous cleanliness and by keeping the skin dry with frequent dressing changes.

29

DEATH IN INTESTINAL

on
the
surgical
result of

Death in intestinal obstruction must be same considered from two points of view first we are consider the factors affecting the mortality may be and second we must consider the obstruction occurs in a of death

deficient physically ex- who for many years has been
standard diet Such people show
FACTORS AFFECTING RATE OF MORTALITY
Access to surgical intervention

Associated Disease

Patients with intestinal obstruction who have associated disease such as cardiac disease coronary disease cirrhosis of the liver or renal disease swell the mortality rate for intestinal obstruction In such cases the involved organs may not be able to carry the additional load thrown upon them by the presence of an obstructing process in the small bowel

Circulatory Changes in the Obstructed Bowel Wall

The circulatory changes that take place in the obstructed bowel wall constitute another important factor in the mortality rate These changes may come about in two ways First there may be a gross interference in the mesenteric circulation and second there may be interference with the capillary circulation of the bowel wall itself due to an increase in the intraluminal pressure Extreme interference with the mesenteric circulation invariably results in gangrene of the bowel and the course of the disease in these cases is fulminating The altered physiologic conditions in strangulating obstructions differ from those in nonstrangulating obstructions It is often difficult to evaluate the degree of capillary interference in the bowel

Many surgeons take little note of this capillary interference except to indicate the presence of it in the peritoneal cavity which in itself is indicative of capillary compression Such capillary compression which is associated with long standing intestinal distention of high degree may cause changes in the bowel wall which can lead to necrosis and perforation This is especially likely to occur in the cecum due to obstruction of the sigmoid

Age

The age of the patient definitely influences the mortality rate in intestinal obstruction It has been amply shown that the mortality rate is very high in patients under the age of one and over the age of seventy In early infancy congenital anomalies plus the inability of such patients to withstand fluid loss and surgical procedures of great magnitude are largely responsible for the increased mortality rate In infants electrolyte imbalances occur very rapidly as a result of obstruction Further more many of these infants present anomalies of such extensiveness that they are not amenable to surgical correction In the aged on the other hand the superimposition of the obstructive process upon an already worn out body mechanism may be the straw that breaks the camel's back

Clinical and Surgical Judgement of Attending Physician

The clinical judgement and diagnostic acumen of the attending physician as well as his surgical ability are important factors affecting the mortality rate This is especially true in those cases of intestinal obstruction that occur within the first week following previous surgery In such cases the clinical picture is frequently complicated by varying degrees of peritoneal irritation or even frank peritonitis as a result of the previous surgical procedure In addition the onset of the obstructive symptoms is often insidious the picture changing almost imperceptibly from that of a stormy post operative convalescence to a full blown intestinal obstruction In such cases the diagnostic acumen of the attending surgeon is strained to the utmost in differentiating those patients with frank intestinal obstruction from those in whom there is

peritoneal infection or in whom there is a post operative ileus as a result of too much or too rough handling of tissues

In the final analysis it is the surgical judgement of the attending man which decides whether the patient requires immediate surgery or whether conservative measures should be instituted at least for the time being. A timid approach by the surgeon is apt to result in a fatality. Boldness must be coupled with sound clinical judgement and the application of well developed surgical principles in order to assure a low mortality. Any further reduction in the mortality rate for intestinal obstruction will be produced by this one factor of good surgical judgement.

CAUSE OF DEATH IN INTESTINAL OBSTRUCTION

The complexity of factors causing death in intestinal obstruction is such that one must divide the causes of death into primary factors (the result of the obstruction itself) and secondary factors (conditions present at the time of the obstruction and in no way connected with the obstructive process).

Primary Causes of Death in Intestinal Obstruction

The primary causes of death may be grouped as follows:

1. Death as a result of changes in the blood chemistry and electrolyte imbalances
2. Death resulting from strangulation or shock
3. Death due to peritonitis caused by leakage or perforation

Death Due to Changes in Blood Chemistry and Electrolyte Imbalances. A vast amount of experimental data has been accumulated and as a result many theories have been evolved in an attempt to explain the lethal mechanism and the pathologic alterations occurring during the course of intestinal obstruction. Much of the recent experimental study on bowel obstruction has emphasized electrolyte imbalance and hemodynamic disturbances as the most important factors. The previously held concept that the lethal factor is the result of absorption of a toxin in the obstructed

bowel has been relegated to a position of secondary importance.

Electrolyte Imbalance. The consensus among most workers at the present time is that death is due both to a marked disturbance of the water and electrolyte balance and to dehydration. The electrolyte imbalance chiefly involves sodium, potassium and chloride. The electrolyte imbalance and dehydration in intestinal obstruction may be caused by (1) vomiting which produces an alkalosis due to chloride loss, (2) continuous suction applied to gastric tube without adequate replacement and (3) loss of fluid and electrolytes into the gastro-intestinal tract as a result of the distention. In sufficient food intake as a result of the obstruction or failure to utilize food that is taken produces starvation. An additional change in blood chemistry is the increase in the nonprotein nitrogen caused by toxic absorption and by a lack of elimination by the patient. Many deaths may be traced directly to these causes. With the exception of high intestinal obstruction, none of these changes is rapid and with the proper administration of water, sodium, chloride, potassium and glucose the patient may be carried over a relatively long period of time. At present it is rather well established that in untreated cases of high intestinal obstruction death is preceded and abetted by the progressive development of severe alkalosis by a lowering of the blood chlorides and at last by an increase in the blood urea. It has also been demonstrated that the administration of both chlorides and water below the point of obstruction causes the chemistry of the blood, the urine and stomach contents to remain normal and also greatly prolongs life, particularly if potassium is added to the intravenous fluid. Death in such cases occurs only after some three or four weeks and cannot be attributed directly to the obstruction. Most surgeons at present believe that death in high intestinal obstruction is not in any way related to toxemia but is due solely to chemical imbalance following the loss of chlorides in water and may be prevented by supplying chlorides intravenously.

Holt measured the amount of fluid lost in strangulated loops of bowel. He concluded from this that in long loop obstruction sufficient fluid may be lost to cause death by dehydration. This

experimental study has been objected to by many workers. Although there is some confusion as to the relative role of fluid loss and strangulation the experimental studies do lead one to believe that with long loop obstructions the loss of fluid in itself may be a contributing factor in the death of the patient.

Plasma Loss. Gendel and Fine studied the effect of acute intestinal obstruction on the blood and plasma volume. They noted that in experimental animals with intestinal obstruction death was more rapid if the bowel were distended than if it were not. In addition, if strangulation of the bowel were superimposed upon distention, death occurred even more rapidly. Distention of the obstructed intestine in experimental animals results in an early and progressive loss of blood plasma. The average loss of plasma reaches 36 per cent within four to six hours and 55 per cent within 24 hours. A 55 per cent loss of plasma is roughly equivalent to 31 per cent of the entire body weight. Of this amount Gendel and Fine believe that 0.7 per cent can be attributed to dehydration and 2.38 per cent to the harmful influence of distention on the general circulation. A loss of 2.38 per cent of the body weight in terms of plasma is believed to be sufficient to cause death. The degree of plasma loss due to distention alone is sufficient to indicate that this may well cause death.

Toxic Factor. Attempts to isolate the toxin or organism responsible for the hypotensive toxemia associated with intestinal obstruction have been carried out recently. Many toxins and organisms, especially *Bacillus botulinus* and the *Bacillus cereus* have been implicated. Conclusive evidence of the presence of the toxin and its isolation in a pure form has not been obtained to date. There is no doubt, however, that bacterial contamination coupled with other factors does play a role in mortality. This can be demonstrated by the protective action of the antibiotic drugs in prolonging life in the obstructed strangulated bowel. This toxin supposedly may be formed either in the mucosa and absorbed directly or formed within the lumen of the intestine as a result of the action of normal digestive secretions, abnormal or perverted secretions or bacteria on the stagnated contents of the obstructed bowel. The absorption of this supposed

toxic substance within the bowel is believed to be influenced by the degree of damage to the mucosa from distention and by the increased intraluminal pressure.

Death Resulting from Strangulation and Shock. The cause of death in strangulating obstruction in experimental animals varies according to the length of the involved loop of bowel and the mechanism of the obstructing process. Acute blood loss may cause death in a long loop obstruction before any toxic changes can occur. A fatal outcome may also occur in short loop strangulating obstructions like those found with hernias because of the rapid death of tissue with perforation and peritonitis. In medium length loops a true toxic substance is believed to be present which may cause death in the absence of any perforation or significant blood loss. In these cases the lethal outcome is believed to be caused by the production of toxin due to the action of proteolytic organisms upon devitalized tissues. The general condition of the patient seems to depend upon the extent of the strangulation and not upon the amount of toxic substance within the bowel. In some cases the strangulation may be so gradual that it does not produce the signs and symptoms of strangulating obstruction. In the acutely strangulated cases the profound toxic changes frequently associated with strangulating obstructions are most likely to occur early. The more extensive the strangulation and the more rapid its onset, the more profound this toxemia may be.

In addition to the foregoing factors responsible for death in intestinal obstruction an additional factor should be mentioned, i.e. the amount of strangulated bowel which requires removal. In some cases resection of this bowel may leave a segment of intestinal tract so small that it is incompatible with life. It has been estimated that 50 per cent of the small bowel is necessary to maintain the proper nutrition. Although successful operations have been reported on strangulating obstructions which require the removal of 70 to 80 per cent of the small bowel eventually result in the death of the patient through inanition.

Strangulation and shock are common causes of death in intestinal obstruction. It has long been known that when a large segment of bowel is sud-

denly and completely strangulated a rapid and severe state of shock may ensue. It has also been shown that where large segments of bowel have become suddenly strangulated the shock may be so severe that it may be the primary cause of death. Although a post mortem in such cases may demonstrate only slight dilatation of the small bowel and no signs of toxemia or peritonitis yet profound prostration and death may occur shortly after surgery as a result of the shock of surgery added to the pre existing condition of the patient. Shock is the second most common cause of death in intestinal obstruction. Patients admitted to the hospital and taken to surgery before dehydration and electrolyte imbalance have been corrected are very likely to go into shock before during or after surgical intervention. Secondary factors which influence the mortality rate by precipitating the shock state are associated diseases such as heart disease renal disease pneumonia and cerebrovascular accidents.

Death Due to Peritonitis. A common cause of death in cases of intestinal obstruction particularly those low in the gastro intestinal tract is peritonitis. This may be caused by necrosis of the bowel wall with perforation leakage of the line of anastomosis or leakage of an improperly performed enterostomy. Although the use of antibiotics has done much to decrease the incidence of this cause of death continuous leakage from the bowel which does not wall off or form a fistula is invariably fatal.

Secondary Causes of Death in Intestinal Obstruction

The disease process *per se* may cause the death of the patient as is the case with metastatic carcinoma for example. Although the obstruction may be relieved the metastasis of the carcinoma may be so extensive that it results in the death of the patient.

Cardiac decompensation and coronary thrombosis particularly in the older age groups are not uncommon causes of death in intestinal obstruction whether the patient is surgically treated or not.

Pulmonary emboli may occur in an occasional case and cause failure in an otherwise successfully

treated surgical patient. Inhalation of vomitus while under anesthesia may occur producing asphyxia and death. Death may occur on the operating table as a result of anesthesia due to a spinal or in some cases the patient may develop cardiac arrest. In addition death on the operating table may occur from shock as a result of failure to put the patient into the proper state of electrolyte balance.

A lower nephron syndrome may occasionally occur with intestinal obstruction which results in a suppression of urine and the death of the patient.

Pneumonia is the principal cause of death in many cases. The onset of pneumonia may be insidious so much so in fact that a well developed full blown pneumonia may be present before the attending surgeon is aware of its presence. The most reliable diagnostic aid is the chest X ray. This is particularly important in the aged or the very young patient.

In an occasional case the cause of death cannot be determined. At autopsy in such patients one may find little or nothing to explain the reason for death.

Additional Controversial Causes of Death

The role of the liver in intestinal obstruction is still controversial. Reports of central necrosis in the mild form have been reported in intestinal obstruction. Hepatic function tests however, show slightly decreased values but at present appear to be insufficient to implicate the liver as a cause of death.

Many laboratory and clinical workers have been impressed by the consistent cellular changes and lipid depletion observed in the adrenal cortex after death in experimental as well as clinical intestinal obstruction. The fact that experimental animals given adrenal cortical extract survived longer than those controlled dogs not so treated seems to be corroborative evidence. It is a well known fact that the adrenal cortex is essential for the performance of certain functions in the control of electrolyte balance. In addition the adrenal cortex is necessary for life. The idea however that terminal collapse in a case of intestinal obstruction may be the result of adrenal cortical failure still remains in the realm of theory.

BIBLIOGRAPHY

- ABBOTT W O Intubation studies of the human small intestine A treatment of intestinal obstruction and a procedure for identifying the lesion Arch Int Med 63 453 1939
- ABBOTT W O Pole of intubation in treatment of intestinal obstruction Ohio M J 36 1001 1940
- ABBOTT W O Indications for the use of the Miller Abbott tube New England J Med 225 641 1941
- ABBOTT W O Intestinal obstruction from practitioner's point of view New York J Med 42 421 1947
- ABBOTT W O AND JOHNSTON C G Intubation studies of the human small intestine Surg Gynec & Obst 66 691 1938
- ABBOTT W O LEVEY S FORSMAN P C KRIEGER H AND HOLDEN W D The danger of administering parenteral fluids by hypodermoclysis Surgery 32 305 1952
- ABBOTT W O AND MELLORS R C Body fluid studies in experimental obstruction at various levels of the gastrointestinal tract Surgery 12 445 1942
- ABBOTT W O MELLORS R C AND MUNTWYLER E Fluid protein and electrolyte alterations in experimental intestinal obstruction Ann Surg 117 39 1943
- ABBOTT W O AND PENDERGRASS E P Intubation studies of human small intestine motor effects of single clinical doses of morphine sulphate in normal subjects Am J Roentgenol 35 289 1936
- ABBOTT W O AND RAWSON A J A tube for use in the postoperative care of gastroenterostomy cases JAMA 108 1875 1937
- ABELL I Acute intestinal obstruction JAMA 95 1903 1930
- ABLE L W Duodenal obstruction in infants Texas J Med 48 748 1952
- ABRAM H S Intussusception particular reference to roentgen diagnosis without opaque media Radiology 36 490 1941
- ACON W C COTTRELL J D AND DAVIS R G Adenocarcinoma of small bowel Canad M A J 67 258 1952
- ADAMS J E Paralytic ileus as a sequel of fractured ribs Ann Surg 51 107 1910
- ADAMS Tr Med Chir 35 57 1852
- ADLER H F ATKINSON A J AND IVA A C Supple-
mentary and synergistic action of stimulating drugs on motility of human colon Surg Gynec & Obst 74 809 1942
- ADOLPH F F Physiological Remittens Jaques Cattell Press Lancaster Pa 1943
- ADMETTA PAULUS Ven Books f Paulus Aegm ta trans by Francis Adams Sydenham Society London 1847
- AGUIAR C DE P United States Patent Office # 2,356 659 Washington DC 1944
- AIRD I Intestinal obstruction Edinburgh M J 43 375 1936
- AIRD I Morbid influences in intestinal obstruction Ann Surg 114 385 1941
- AIRD L AND HENDERSON W K Intestinal obstruction Lancet 2 373 1936
- AKERS D R Congenital atresia and stenosis of the small intestine Rocky Mountain M J 49 841 1952
- ALBERS J H AND SMITH L L A comparison of cecostomy and transverse colostomy in complete colon obstruction Surg Gynec & Obst 95 410 1952
- ALBITSKY cited by SNOOPGRASS T J Acute intestinal obstruction caused by non absorbable suture material Surgery 6 437 1939
- ALDRICH J F AND BARNES B S Obstruction of the small intestine due to gallstones J Iowa M Soc 23 82 1933
- ALEMAN S Jejuno gastric intussusception rare complication of stomach operation Acta radiol 29 383 1948
- ALBEE R H AND MAYFIELD J L Obstruction of duodenum in case of failure of rotation of intestine as associated with unfused pancreas and mesenteric bands Mil Surgeon 103 452 1948
- ALLEN A W Surgery of diverticulitis of the colon Am J Surg 86 545 1953
- ALLEN R F A study of the effect of terramycin on intestinal flora Am J Surg 86 678 1953
- ALLINGHAM W Diseases of Rectum 4th ed The Blakiston Co Philadelphia 1882
- ALTEMEIER J A AND WADSWORTH G H Ileus following fractured ribs Ann Surg 115 32 1942
- ALVAREZ W C The danger and folly of giving a barium meal to patients with intestinal obstruction Am J Digest Dis 6 476 1938
- ALVAREZ W C Hysterical type of non gaseous abdominal bloating Arch Int Med 84 217 1949
- ALVAREZ W C AND HOSOF J What has happened to the unobstructed bowel that fails to transport fluids and gas? Amer J Surg 6 569 1929
- AMENIOLA F H Gastroduodenal intussusception Ann Surg 178 1028 1948
- AMUSSAT J Z Memoire sur la Possibilite d'Etudier un Anus Artificiel dans la Region Lombaire sans Penetrer dans le Peritone Paris 1839
- AMUSSAT J Z Relation de la Maladie de Broussais Suru d Quelques Reflexions Pratiques sur les Obstructions du Rectum Malteste Paris 1839
- AMUSSAT J Z Anus artificiel Bull Acad nat med Paris 4 218 1839-40

- ANDERS J M Stenosis of the duodenum a statistical study with report of new case Am J M Sc 144 360 1912
- ANDERSON A L AND ANDERSON C W Intestinal obstruction due to a watermelon seed New Orleans M & S J 83 256 1930
- ANDERSON D H AND DOOB E F Leiomyosarcoma of duodenum Arch Path 16 795 1933
- ANDERSON J R COUNSELLER V S AND WOOLNER L B Lithopedion report of an unusual case and notes of two other cases Amer J Obst & Gynec 62 439 1951
- ANDERSON K AND RINGSTED A Analysis of gas in intestinal tract Acta chir scandinav 88 475 1943
- ANDERSON R E AND TANTURI C A Identification of the toxin of clostridium organisms in experimental intestinal obstruction Arch Surg 60 143 1950
- ANDREWS W Duodenal hernia a misnomer Surg Gynec & Obst 37 740 1923
- ANGLE L W Acute intestinal obstruction caused by impacted gallstones Am J Surg 17 364 1932
- ANTONCIC R F AND LAWSON H The muscular activity of the small intestine in the dog during acute obstruction Ann Surg 114 415 1941
- ANTONCIC R F AND LAWSON H The neurogenic factor in intestinal obstruction Surg Gynec & Obst 72 728 1941
- APLEY A G Intussusception due to sarcoma Lancet 2 314 1939
- APSYRTUS Cited in Ciba Symposia Vol 5 1944
- ARIEL I M The effects of acute hypochloremia on the distribution of body fluid and composition of tissue electrolytes in man Ann Surg 140 150 1954
- ARNITAGE G Intestinal obstruction complicating posterior gastrojejunostomy Case of internal strangulation of small intestine by afferent limb Brit J Surg 18 154 1930
- ARMOUR J C BROWN T G DUNLOP D M MITCHELL T G SEARSL H H AND STEWART C P Studies on high intestinal obstruction Brit J Surg 18 467 1931
- ARNHEIM F F Congenital ileal atresia with gangrene perforation and peritonitis in a newborn infant Am J Dis Child 69 108 1945
- ARNOLD D G Intestinal obstruction caused by gallstones Am J Surg 52 381 1941
- ARNOLD I A Hernia into the paraduodenal fossa with obstruction due to a large biliary calculus JAMA 112 1245 1939
- ASCROFT P B AND SAMUEL E Radiography in postoperative ileus Lancet 2 445 1940
- ASCROFT P B AND SAMUEL E X ray diagnosis of acute intestinal obstruction Brit J Radiol 14 11 1941
- ASHURST J JR The Intestinal Encyclopaedia of Surgery Wm Wood and Co New York 1886
- ASTICALL A Intestinal obstruction following operations on the lower part of the abdomen M J Australia 2 713 1933
- BADIA P D Acute intussusception M Jme 70 373 1942
- BAEYER E V Diagnosis of congenital obstruction of the stomach and small intestine in the newborn Radiology 52 157 1949
- BAILEY F W Surgery of the aged Am J Surg 24 487 1934
- BAILLIE M Anatomie des drankhaften Baues von Eingeweiden der Wichtigsten theile im menschlichen Korper Aus dem Engl mit Zusatz von S Th Sommering Voss Berlin 1794
- BAIRD L W AND KIRKLIN B R Obstruction of the third portion of the duodenum of unusual etiology Radiology 27 235 1936
- BAKER C R F Treatment of mechanical intestinal obstruction by tube and suction J South Carolina M A 33 53 1937
- BALANGERO F I Postoperative intestinal obstruction J Internat Coll Surgeons 18 737 1952
- BALCH F G JR Gallstone ileus New England J Med 21 457 1938
- BALDY cited by RICHARDSON E P Intestinal obstruction following Baldy Webster operation Surg Gynec & Obst 31 90 1920
- BALFOUR D C Primary retrograde intussusception of the sigmoid associated with tumor Ann Surg 68 588 1918
- BALFOUR D C BOLLMAN J L AND GRINDLAY J H Intestinal lymph flow following simple intestinal obstruction in the rat Surgery 29 500 1951
- BANCROFT F W Surgery in the aged New York J Med 43 37 1943
- BANERJI B N Volvulus of the caecum Indian J Surg 13 311 1951
- BANNER E A HUNT A B AND DIXON C F Pregnancy associated with carcinoma of the large intestine Surg Gynec & Obst 80 211 1945
- BARDER W H Dilatation of the duodenum an experimental study Ann Surg 62 433 1915
- BARRETT P cited by TREMPER F Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment Wm Wood and Co New York 1899
- BARDOSA J J DE CASTRO Volvulus of the entire small intestine and its mesentery Am J Surg 64 400 1944
- BARGEN J A ADSON A W LUNDY J S AND DIXON C F Functional abdominal distention simulating megacolon Am J Digest Dis 3 17 1936
- BARKER A E A suggestion for the treatment of irreducible intussusception of the bowel Lancet 1 79 1892
- BARNES B C Acute small bowel obstruction J Iowa Med Soc 41 43 1951
- BARNES F L What minimum symptomatology justifies a clinical diagnosis of acute intestinal obstruction? M Rec & Ann 31 419 1937
- BARNF J P Surgical treatment of irreducible intussusception in infants Surg Gynec & Obst 83 741 1947
- BARNES J I Left sided approach for small bowel obstructions Texas J Med 45 281 1944

- BARRETT J M Reducing the hazards of acute intestinal obstruction An address delivered before the 33th annual session of the Chattahoochee Valley Medical and Surgical Association pp 1-6, 1935
- BARRETT C O AND GALE K I Mechanical intestinal obstruction *Am J Surg* 86 23, 1953
- BARONOFFSKY I D Primary resection and aseptic end to end anastomosis for acute or subacute large bowel obstruction *Surgery* 27 664 1950
- BARONOFFSKY I D Primary end to end anastomosis for congenital atresia of the small bowel *Surgery* 30 841 1951
- PARRY H C Fibrous stricture of the small intestine following strangulated hernia *Brit J Surg* 30 64 1942
- BARTHOLIN cited by MARTIN F Intestinal obstruction due to gallstones *Ann Surg* 55 725 1912
- BARTLETT W JR An indication for early operation in intestinal obstruction *Surg Gynec & Obst* 49 719 1929
- BASSLER A A cause of peritoneal adhesions in a new bacillus the *Bacillus adhesioformis* *M Rec* 85 427 1914
- BAUDAMANT Description de deux cas masses de cheveux trouvees dans l'estomac et les intestins d'un jeune garçon age de seize ans *Hist soc roy de med Paris* 2 262 1777
- BAUMGARTNER C J Intestinal obstruction *M Arts & Sc* 1 45 1947
- BAUMGARTNER C J Survey of intestinal obstruction *Arch Surg* 55 607 1947
- BAUMGARTNER C J Intestinal obstruction *Los Angeles Surg Soc* 1 24 1948
- BAUMGARTNER W Invagination de abführenden Jejunumschlinge in die braunschweigische Anastomose nach vor derer Billroth II Resektion *Zentralbl Chir* 65 1907 1938
- PEARM S F Gallstone ileus *J Misouri M A* 35 489 1933
- PEARSON J M Cecostomy *Am J Surg* 84 236 1952
- PELAFRÈRE J V The lure of medical history pharmacy of the ancient Egyptians *Calif & West Med* 31 47 1959
- BECKER W F Acute adhesive ileus *Surg Gynec & Obst* 95 472 1952
- PECKER W F Acute obstruction of the colon *Surg Gynec & Obst* 96 677 1953
- BECKER W F DAVIS C E JR AND LEHMAN F Intestinal obstruction *Ann. Surg* 131 385 1950
- BEDNFIELD H A review of visceroproposis and allied abdominal conditions associated with chronic invalidism *Quart J Med* 22 611 1928-1929
- BEHRE, M R Jejunal polyposis with intussusception and melanin spots *J Pediatr* 38 641 1951
- BEING C A BOSCH D T AND CARTER, O P JR Blood volume in geriatric surgery *Geriatrics* 7 179 1952
- PERI R The medicine man or Indian and Eskimo notions of medicine *Canad J M Sc* 14 456 1885-1886
- BELLINGHAM F MACKAY R AND WINSTON C Pregnancy and intestinal obstruction a dangerous combination *Med J Australia* 2 318 1949
- BELLIS C J, AND LARSON W P The impermeability of the viable obstructed bowel of dogs to *Clostridium botulinum* toxin *Surgery* 6 901 1939
- BELLIS C J AND WANGENSTEEN O H Venous circulatory changes in the abdomen and lower extremities attending intestinal distention *Proc. Soc Exper Biol & Med*, 41 490 1939
- BENEFIT E B STEWART C P AND CUTNER P N The role of bile in high intestinal obstruction *Surg Gynec & Obst*, 54 605 1932
- BENEDICT F G A study of prolonged fasting Publication # 203 Carnegie Institution of Washington Washington DC 1915
- BENKE cited by BENZNER A G Extensive resection of the small intestine *Ann. Surg* 89 675 1929
- BENVENI ANTONIO *De Abditis Annulis ad Mirandis Morborum et Sanationum Causis* trans by Charles Singer Charles C Thomas Co Springfield Ill 1954
- BENJAMIN A F Volvulus of the colon *Minnesota Med*, 22 874 1939
- BENJAMIN A F Acute obstruction of the bowel *Minnesota Med* 26 1009 1943
- BENNETT C Obstruction of small intestine by gallstones *Brit M J* 1 565 1976
- BENNETT H M Atresia of the small intestine *Brit M J*, 2 1369 1950
- BENNETT L C Diagnosis and treatment of obstruction of the small intestine *Am J Surg* 62 59 1943
- BENNETT L C Intubation management of distention in intestinal obstruction *West J Surg*, 49 71 1944
- BENSON C D AND COLRY J J Congenital intrinsic obstruction of the stomach and duodenum in the newborn *Arch Surg*, 62 856 1951
- BENSON C D AND FENBERG G C Congenital duodenal obstruction (intrinsic obstruction) *Arch Surg*, 56 58 1948
- BERG A A *Surgical Diagnosis a Manual for Students and Practitioners* Lea Brothers New York 1905
- PERGER, K E AND LINDERER E A Intestinal volvulus precipitated by lead poisoning *JAMA* 147 13 1951
- BERGER I AND ACHS S Perforation of the small intestine by the Miller Abbott tube *Surgery* 22 648 1947
- BERLINER, R W KENNEDY T J, AND HILTON J G Renal mechanisms for the excretion of potassium *Am J Physiol* 162 348 1950
- BERLINER R W, KENNEDY T J AND ORLOFF J Relationship between acidification of the urine and potassium metabolism Effect of carbonic anhydrase inhibition on potassium excretion *Am J Med* 11 274 1951
- PERRAY P Duodenal tube—simple technique of rapid tubage *Lyon med* 152 717 1933

- BERNAY P Instantaneous tubage with Camus sound technique and results after 90 soundings Lyon med 153 447 1934
- BERNHIM F AND BERNHIM M Action of drugs on choline esterase of brain J Pharmacol & Exper Therap 57 427 1936
- BERRY R E L Diagnosis and treatment of acute intestinal obstruction JAMA 148 347 1952
- BERRY R E L Abstracts of cases previously reported Clinical aspects of acute peripheral oligemia from plasma water and sodium salt loss Arch Surg 67 408 1954
- BERRY R E L IOB V AND CAMPBELL K N Potassium metabolism in the immediate postoperative period Arch Surg 57 470 1948
- BERRY R E L IOB V AND HODGSON P Tolerance of elderly surgical patients to intravenous dextrose and water solutions Arch Surg 69 315 1954
- BERTI cited by SINGLETON A C Chronic gastric volvulus Radiology 34 53 1940
- BESSER E L Cause of death in cases of mechanical intestinal obstruction Arch Surg 41 970 1940
- RESE C H AND TAYLOR W B *The Physiological Basis of Medical Practice* 6th ed The Williams and Wilkins Co Baltimore 1935
- PICKHAM W S *Operative Surgery* vol 4 p 942 W B Saunders Co Philadelphia 1924
- BILLINGS F T Knotting of duodenal tube in situ JAMA 80 1774 1923
- BINNINGER J N *Observationum et Curationum Medicinalium Centuria Quinque* Oberv 81 p 222 Mont belgardii Hyppionis 1673
- BIRNBAUM W Inflammation of the vermiform appendix by metallic mercury Am J Surg 74 494 1947
- BISGARD J D AND JOHNSON E K Influence of certain drugs and anesthetics upon gastrointestinal tone and motility Ann Surg 110 802 1939
- BISGARD J D AND NYE D Influence of hot and cold application upon gastric and intestinal motor activity Surg Cynece & Obst 71 172 1940
- BISHOP L H JR Congenital anomalies of the lower gastrointestinal tract causing obstruction I M A Georgia 36 223 1947
- BISHOP W A Intussusception of the vermiform appendix The intussusception protruding from the anus Brit J Surg 1 721 1913
- BLACKBURN C R B HENSELY D E G, AND WRIGHT F B Studies on intravascular hemolysis in man The pathogenesis of the initial stages of acute renal failure J Clin Invest 33 825 1954
- BLACKBURN J H Clinical and surgical aspects of acute intestinal obstruction Kentucky M J 30 242 1932
- BLAIN A III Penicillin in experimental intestinal obstruction Surg Cynece & Obst 84 753 1947
- BLAIN A III AND HARRIS H N Intestinal obstruction due to perforations of the gallbladder Surgery 21 110 1947
- BLAIN A III KENNEDY J D CALHAN R J AND HARRIS H N Effect of penicillin experimental intestinal obstruction Arch Surg 53 378 1946
- BLAKELY S B Abdominal pain in pregnancy JAMA 101 970 1933
- BLACKLOCK A Decompression of the intestinal tract Surg Gynece & Obst 68 842 1939
- BLACKLOCK T T The importance of the Miller Abbott tube in intestinal obstruction U S Naval M Bull 40 376, 1942
- BLAND J H *The Clinical Use of Fluid and Electrolyte* W B Saunders Co Philadelphia 1952
- BLAND J H Regulation of fluid and electrolyte balance in aged persons J Am Geriatrics Soc 1 233 1953
- BLAYLAND A J Fibroid myoma of the stomach Brit J Surg 19 339 1931
- BLISS R W Acute intestinal obstruction Mil Surgeon 71 152 1932
- BLISS T Intussusception after appendectomy Brit M J 2 405 1944
- BLOCH W Antiperistaltik des Dickdarms beim Menschen Med klin 7 219 1911
- BLOCK F B Intestinal obstruction caused by food Am J M Sc 185 356 1933
- BLODGETT J B A technique for the satisfactory use of the Miller Abbott tube Am J Surg 53 271 1941
- BLODGETT J B An evaluation of intestinal suction in intestinal obstruction Surgery 11 739 1942
- BLOODGOOD J C Acute dilatation of stomach Ann Surg 46 736 1907
- BLOODGOOD J C Intestinal obstruction due to volvulus or adhesions of sigmoid colon with a report of five cases and a study of etiological factors Ann Surg 49 161 1909
- BLOODGOOD J C Dilatation of the duodenum in relation to surgery of the stomach and colon JAMA 59 117 1912
- BLOOM B M DORTCH H JR LEWIN T H KIBLER A F AND SHEPARD K S The effect of heparin upon intra abdominal adhesions in rabbits Ann Surg 176 324 1947
- ROCKUS H L *Castro enterology* W P Saunders Co Philadelphia 1946
- BODENHEIMER M CASTEN D AND FRIED J J Acute intestinal obstruction J Internat Coll Surgeons 3 28 1940
- BODENHEIMER M CASTEN D AND FRIED J J A study of the modern methods of the treatment of intestinal obstruction J Internat Coll Surgeons 10 174 1947
- BODIAN M WHITE, L L R CARTER C O, AND LOUW J H Congenital duodenal obstruction and mongolism Brit M J 1 77 1952
- BORART A H The surgical significance of intestinal angioneurotic edema with an illustrative case Ann Surg 61 374 1915
- BOLINGER J A AND FOWLER F F Results of treatment of acute small bowel obstruction Arch Surg 66 888 1951

- BOYLE S AND TILLY I Direct sounding of duodenum by means of a metal mandrin and with aid of roentgen illumination *Munchen med Wchnschr* 69 1573 1922 abstract J A M A 80 218 1923
- BOYFUS cited by TREVES F *Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- BOYSE V On operative paralytic distention of intestine with special reference to its treatment by jejunostomy *Arch Middlesex Hosp* 21 39 1910
- BOYSE V Functional derangement of intestine that follows abdominal operations *Lancet* 2 1373 1934
- BOYSE V Functional intestinal obstruction without obvious antecedent cause *Brit J Surg* 40 78 1952
- BOYMAN C N AND RIGLER L G Spontaneous internal biliary fistula and gallstone obstruction *Surgery* 1 349 1937
- BOWEN B Volvulus operation and recovery J A M A 108 43 1937
- BOUTEON R PIFTRI H SIROT L AND STOPPA In vaginotomy tardive a travers une bouche de gastrectomie Afrique Fr *Chir* No 3-4 p 68-70 1952
- BOUYERET L La tension intermittente de l'epigastre *Lyon medicale* 96 465 1901
- BOWEN F H Intussusception associated with a polyp in a Meckel's diverticulum J M A Georgia 30 390 1941
- BOWERS W F AND TITRUP L Congenital intestinal atresia *Staff Meet Bull Hosp of Univ Minn* 8 280 1937
- BOYCE F F AND Mc FETTRIDGE E M Acute intestinal obstruction *South Surgeon* 6 109 1937
- BOYD R Description of a malformation of duodenum *Lancet* 1 648 1845
- BOYS F Prophylaxis of peritoneal adhesions review of literature *Surgery* 11 118 1942
- BRAMBRIDGE Personal communication cited by KERR W G AND KIRKCALDY WILLIS W H Volvulus of the small intestine *Brit M J* 1 799 1946
- BRAINE J Cancer de l'ampoule rectale coïncidant avec un endométriose recto sigmoidien ténosant développé dans la paroi même du rectum Amputation abdomino-perinéale datant de 14 mois *Arch mal app digest* 22 673 1932
- BRANDBERG R An experimental study of intestinal motility in mechanical ileus *Acta chir scandinav* 83 287 1939
- BRASH J C AND JAMESON E B *Cunningham's Manual of Practical Anatomy* p 333 Oxford University Press New York 1935
- BRAUN cited by TREVES F *Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- BRAUN H Ueber Gastroenterostomie und Gleichzeitig ausgeführte Enteronastomose *Arch klin Chir* 45 361 1892
- BRAYTON D AND NORRI W J Intussusception in infants *Am J Surg* 88 37 1952
- BRENIZER A G Deformities and obstruction of the duodenum *Am J Surg* 9 430 1930
- BRENIZER A G Extensive resections of the small intestine *Ann Surg* 93 788 1931
- BRENIZER A G Unusual complication of intestinal intubation *New England J Med* 238 291 1948
- BRESCHET G cited by THOMPSON P A case of polypus of the pylorus with intussusception J Anat & Physiol 31 392 1897
- BREUER B Ueber ein neues Roentgensymptom der Gallensteinkrankheit *Roentgenpraxis* 3 879 1931
- BRINDLEY G V Acute obstruction of the colon *Surg Gynec & Obst* 78 556 1944
- BRINDLEY G V Acute obstructions of the colon *Texas J Med* 40 571 1945
- BRINTON W L *Intestinal Obstruction* John Churchill and Sons London 1867
- BROQUÉ P AND FUEHL L Intubation de l'intestin grêle et du colon par sonde à lumière unique et lestée au mercure *Mém Acad chir Paris* 75 5 1949
- BROWN W Herniation and exstrophy of the ileum in the newborn incidental to adherent Meckel's diverticulum in the umbilical area *West Virginia M J* 43 120 1947
- BROOKSHER W R, JR The x-ray diagnosis of acute intestinal obstruction *Internat J Med & Surg* 45 416 1932
- BROWN H P JR Intra peritoneal hernia of the ileum through a rent in mesentery *Ann Surg* 72 516 1920
- BROWN J S AND TROWELL H C Deficiency bowel pattern in Polish refugees African and Indian adults and children *Lancet* 2 812 1944
- BROWN R B AND ROSS D Congenital abnormalities of intestinal rotation and mesenteric attachment—a cause of intestinal obstruction in the adult *Ann Surg* 134 88 1951
- BROWN S Gastro intestinal obstructions *Radiology* 15 364 1930
- BROWNE D Neo-natal intestinal obstruction *Proc Roy Soc Med* 44 623 1951
- BRUNN H AND LEVITIN J A roentgenological study of intestinal obstruction *Surg Gynec & Obst* 70 914 1940
- BRUSGAARD C Volvulus of the sigmoid colon and its treatment *Surgery* 22 466 1947
- BRUNER A AND HODGSON J P Intestinal obstruction in fibrocystic disease of the pancreas *Am J Roentgenol* 69 14 1953
- BRYANT J Visceral adhesions and bands normal incidence *Am J M Sc* 163 75 1922
- BUCHANAN G Malformation of duodenum in children *Tr Path Soc London* 12 121 1816
- BUCKSTEIN J *The Digestive Tract in Roentgenology* J B Lippincott Co Philadelphia 1948
- BUCKSTEIN J AND MICHAELS L The roentgen diagnosis of acute intestinal obstruction *Am J Digest Dis* 3 767 1938

- BUCKWALTER J A AND MODLIN M Acute appendicitis with intestinal obstruction JAMA 155 1577 1954
- BUIRGE, R E DENNIS C VARCO R L AND WANGENSTEEN O H Histology of experimental appendical obstruction (rabbit ape and man) Arch Path 30 461 1940
- BUNCH G H AND DOUGHTY R G Chronic obstruction of the proximal duodenum by congenital bands Am J Surg 111 759 1940
- BURGET G E MARTZLOFF K SUCKOW G AND THORNTON C B Closed intestinal loop relation of intraloop pressure to clinical condition of animal Arch Surg 21 829 1930
- BURGHARD F F Intussusception of the vermiform appendix the intussusception protruding from the anus Brit J Surg 1 721 1913
- BURKE J MANN I S AND KIRSH I I Long standing asymptomatic partial intestinal obstruction caused by foreign body and stricture of the ileum Surgery 27 902 1950
- BURKE M S AND WILLIAMS G R Intussusception of small gut due to a foreign body Brit M J 1 394 1944
- BURRITT C Obstruction of small intestine by gallstones Brit M J 1 565 1926
- BURNETT W E ROSEMOND G P AND BUCHER, R M Mesenteric cysts report of 3 cases in one of which calcified cyst was present Arch Surg 60 699 1950
- BURNHAM P J Acute bowel obstruction Arch Surg 66 167 1953
- BURSTEIN C L Effect of cyclopropane on intestinal activity in vivo Proc Soc Exp Biol & Med 38 530 1938
- BURT L I Observations on biliary calculus ulcerating into small intestine M J Australia 1 444 1933
- BUSSE A Ein grosses Neurom gangliocellare des Nervus sympathicus Arch path Anat Berlin 151 66 1898
- BUTTON L L Acute intestinal obstruction analysis of Wellington Hospital cases and statistics New Zealand M J 32 320 1933
- CABOT CASE #16292 Recurrent colicky pain chills and abdominal distention New England J Med 203 130 1930
- CABOT CASE #24522 Intestinal obstruction of splenic flexure of colon by fibrous adhesive bands New England J Med 219 1048 1938
- CABOT CASE #27082 Intestinal obstruction due to gallstone New England J Med 224 336 1941
- CAHILL J A JR Obstruction of the sigmoid flexure by a large gallstone Am J Surg 52 285 1941
- CAHN A Heilung von Ileus durch Magenaus spulung, Berl klin Wchnschr 21 669 1884
- CAIRNS J Two examples of children born with preternatural constrictions of the guts Med Essays and Ollet Soc Edinburgh 1 203 1733
- CALIHAN K J KENNEDY I D AND LEVIN A III Intestinal obstruction study of two hundred and four acute cases with reference to the possible efficacy of anti bacterial therapy Bull Johns Hopkins Hosp 79 21 1946
- CALIHAN W A AND HULBERT H F Intestinal obstruction due to gallstones J Internat Coll Surgeons 16 325 1951
- CALISSEN AND AMUSSAT Cited by TREVELL F Intestinal Obstruction 1 aetiology and Their Pathology Diagnosis and Treatment Wm Wood and Co New York 1899
- CAMERON D J Bowel obstruction due to gallstones with report of 3 cases J Indiana M A 30 231 1937
- CAMBELL, A A AND SMITH R G The diagnosis and treatment of volvulus of the sigmoid colon S Clin North America 30 603 1950
- CAMPBELL, S J Mesenteric thrombosis massive resection of small bowel with recovery J M A Alabama 17 304 1948
- CAMUS L Rapid method of duodenal catheterization applied to duodenal intubation Acad de Med Paris 109 388 1933
- CAMUS I Rapid duodenal catheterization and apparatus suggested by Schlumberger Arch mal app digest 28 949 1938
- CAMUS L AND LEVASOR Rapid duodenal tube under roentgen control Presse med 41 1116 1933
- CANNON W B The passage of different food stuffs from the stomach and through the small intestine Am J Physiol 12 387 1904
- CANTOR M O New simplified intestinal decompression tube Am J Surg 72 137 1946
- CANTOR M O Mercury—its role in intestinal decompression tubes Am J Surg 73 690 1947
- CANTOR M O Intestinal Intubation Charles C Thomas Co Springfield Ill 1949
- CANTOR M O Balloons of intestinal decompression tubes trapped in the gastrointestinal tract Grace Hosp Bull pp 51-62 January 1949
- CANTOR, M O The ligament of Treitz as a barrier to intestinal intubation Surgery 26 673 1949
- CANTOR M O Digestive tract decompression Medical Physics 2 274 1950
- CANTOR, M O Effect of variations in the amount of mercury on the speed of intestinal intubation Arch Surg 60 762 1950
- CANTOR M O Physics in intestinal intubation Am J Digest Dis 17 10 1950
- CANTOR M O Radiological criteria for removal of intestinal decompression tube Radiology 54 535 1950
- CANTOR M O Mercury lost in the gastrointestinal tract JAMA 146 560 1951
- CANTOR M O Swallowed intestinal decompression tubes Am J Digest Dis 18 250 1951
- CANTOR M O Intestinal obstruction—1900 vs 1950 J Mich M Soc 52 1194 1951
- CANTOR M O Simplified intestinal decompression Am J Surg 87 122 1954

- CANTOR M O ACKER I SCHIAR A AND FOSTER K
Effect of variation in length of decompression tube upon
bowel wall Am J Surg 82 697 1951
- CANTOR M O KENNEDY C S AND REYNOLDS I P
Use and abuse of intestinal decompression tubes Am
J Surg 73 437 1947
- CANTOR M O MCCOLLUM B F AND HODGES J
Intestinal intubation for barium produced bowel ob-
struction Am J Digest Dis 19 148 1952
- CANTOR M O AND MCGINNIS H P Observations on
effect of drugs upon intestinal intubation Gastroenter-
ology 19 516 1951
- CANTOR M O AND McLEAN D W Intestinal gas
patterns as a diagnostic aid Am J Roentgenol 64
929 1950
- CANTOR M O AND IHELL L P Further simplifying
intestinal intubation Am J Surg 83 516 1952
- CANTOR M O PHELPS I R AND ESLING P H
Effect of hydrogen sulfide upon the balloons of in-
testinal decompression tubes Am J Surg 76 364
1948
- CANTOR IHELPS I R AND ESLING P H Effect of
intestinal gases upon balloons of intestinal decom-
pression tubes Am J Surg 75 441 1948
- CANTOR M O PHELPS E R AND ESLING R H Does
the bowel prevent the outward diffusion of gas from
within the balloon of the intestinal tubes Am J Surg
77 582 1949
- CANTOR, M O AND VANDENBERG H Cecal volvulus in
pregnancy Unpublished data
- CARLSON D J *The Control of Hunger in Health and
Disease* The University of Chicago Press Chicago
1916
- CARLSON H A DVORAK H J LYNCH F W BORMAN
C AND WANGENSTEEN O H Value of X ray evi-
dence of bowel obstruction in various states of intestinal
stasis Proc Soc Exper Biol & Med 28 343 1930
- CARLSON H A AND WANGENSTEEN O H Motor ac-
tivity of the distal bowel in intestinal obstruction com-
parison with the obstructed and normal Proc Soc
Exper Biol & Med 27 676 1930
- CARMICHAEL J L AND GUFFY J L Intestinal obstruc-
tion treatment by reinforced siphonage Am J Surg
31 496 1936
- CARP L Foreign bodies in the intestine Ann Surg 85
575 1927
- CARP I The causes and possible reduction of operative
mortality in geriatric surgery with an analysis of 100
consecutive autopsies records Internat Abstr Surg 87
1 1948
- CARRINGTON G L Safety valve anastomosis and decom-
pression in intestinal surgery by use of a T tube
South Surgeon 11 794 1942
- CARTER F F Congenital occlusion of the duodenum
and small intestine a clinical consideration with report
of two successful cases J Pediatr 2 27 1933
- CARULLA J J Mesenteric vascular occlusion Am J
Surg 85 47 1953
- CARULLA J J Distensiones gaseosas gastro colonicas
y su terapeutica Prensa med argent 31 633 1944
- CARULLA J F Las enterocolitis fermentativas Prensa
med argent 31 1295 1944
- CAROLO J F An unusual complication of Miller Abbott
intubation New England J Med 239 396 1948
- CASE J T X ray Examination of the Alimentary Tract
The Southworth Co Troy N Y 1914
- CASE, J T The value of X ray study in acute bowel
obstruction Illinois M J, 74 326 1938
- CASF J T Umbrathor as a substitute for barium in the
roentgen study of acute intestinal obstruction Am J
Roentgenol 58 422 1947
- CATTS B B Management of acute obstruction of the
small intestines Internat J Med & Surg 46 116 1933
- CATHCART D F Congenital intestinal anomaly in a
new born infant Am J Dis Child 55 566 1938
- CATTELL I B Endometriosis of the colon and rectum
with intestinal obstruction New England J Med 217
9 1937
- CAYLOR H D AND NICKEL A C Intestinal obstruction
from a blood bolus Ann Surg 104 151 1936
- CELSUS D *Medicina* Book 4 trans by W G Spencer
Wm Heinemann Ltd London 1935
- CHAFFEE J S Complications of gastro intestinal intubation
Ann Surg 130 113 1949
- CHAFFIN L MASON B R AND SLEMMONS J M
Intussusception during pregnancy Surg Gynec &
Obst 64 811 1937
- CHALFANT S S Torsion of the caecum with review of
the literature Am J Obst & Gynec 2 597 1921
- CHAMBERLAIN J W Acute intestinal obstruction follow-
ing hernia into ascending mesocolon New England J
Med 216 299 1937
- CHAMBERLAIN W E Roentgenologic aids in the diag-
nosis and management of intestinal obstruction S Clin
North America 18 1621 1938
- CHAPMAN W P FRENCH A B HOFFMAN P S AND
JONES C M Multiple balloon kymograph recording
of the effect of Banthine belladonna and placebos on
upper intestinal motility New England J Med 246
535 1952
- CHAPMAN W P ROWLANDS E N AND JONES C M
Multiple balloon kymographic recording of the com-
parative action of oral administration of atropine tim-
ture of belladonna and placebos on the motility of the
upper small intestine in man New England J Med
243 1 1950
- CHAI IN J L AND STATTERY L R Jejunal stricture
due to ingestion of ammonia JAMA 152 134 1953
- CHAUNCEY I R AND GRAY H K Relationship of
concentration of proteins in serum to postoperative gas-
tric retention Gastroenterology 1 72 1943
- CHEEVER D Evacuation of the small intestine in para-
lytic ileus New England J Med 207 1125 1937
- CHIFFERMAN J T Tw effects of retrograde venous
thrombosis on intestinal strangulation J Pathol & Pract
56 442 1944

- CHESTERMAN J T AND SHEEHAN W J Prophylaxis of paralytic ileus by administration of morphine Brit M J 2 528 1945
- CHLARI H Ueber Intussusception am Magen, Prag med Wchnschr 13 221 1888
- CHIDESTER W C High intestinal obstruction A problem in diagnosis S Clin North America 10 1001 1930
- CHILDS S B AND FOSTER J M The plication operation for intestinal obstruction Rocky Mountain M J 47 917 1950
- CHIZZOLA C Sull importanza del controllo radiologica nel sondaggio duodenale Minerva med 1 918 1930
- CHODOFF cited by LINDENMUTH W W Fecal fistula due to metallic mercury from Miller Abbott tube JAMA 141 986 1949
- CHRISTENSEN H N WILBER P B COYNE, B A AND FISHER J H Effects of simultaneous or prior in fusion of sugars on the fate of infused protein hydrolyates J Clin Invest 34 86 1955
- CHRISTIANSON H W AND BARGEN J A Functional abdominal distention simulating intestinal obstruction Mayo Clin Proc Staff Meet 6 441 1931
- CHRISTOPHER F Benign obstruction of the sigmoid S Clin North America 10 343 1930
- CHRISTOPHER, F Intestinal obstruction complicating acute appendicitis in a child of three years Ann Surg 93 1109 1931
- CHRISTOPHER, F Intestinal obstruction Illinois M J 67 515 1935
- CLAGETT O T MOERSCH H J AND FISCHER A Esophagogastrostomy in the treatment of cardiospasm Surg Gynec & Obst 81 440 1945
- CLARK ALONZO cited by DRAGSTEDT C A Some pharmacological considerations of intestinal obstruction Illinois M J 74 313 1938
- CLARK J H NELSON W LYONS C MAYERSON H S AND DE CAMP P Chronic shock The problem of reduced blood volume in the chronically ill patient Ann Surg 125 618 1947
- CLARKE, J M Treatment of paralytic ileus New Zealand M J 37 193 1938
- CLARKE, J S OBERHELMAN H A EVANS S O AND DRAGSTEDT L R The relation of gastric secretion to events following pyloric obstruction Ann Surg 135 433 1952
- CLOSMADENC cited by GOULD G M AND PYLE, W L Anomalies and Curiosities of Medicine Sydenham Publishers New York 1937
- CLUBBE C P B The Diagnosis and Treatment of Intussusception & J Pentland Edinburgh 1907
- CLUBBE C P B The Diagnosis and Treatment of Intussusception 2nd ed Oxford University Press London 1921
- COCKETT F B A case of acute insufficiency of small intestine Brit J Surg 37 314 1950
- CODINGTON H A Diagnosis and treatment of acute intestinal obstruction South Med & Surg 93 877 1931
- COHEN O H AND SILVERSTEIN M E Unusual complication of Miller Abbott intubation J M Soc New Jersey 49 435 1952
- COHN I JR Progress in study of strangulation intestinal obstruction Am Surgeon 20 1162 1954
- COHN I JR AND HAWTHORNE, H R The role of Clostridium welchii in strangulation obstruction Ann Surg 134 999 1951
- COHN S AND FELMUS R D Volvulus with partial intestinal obstruction caused by an abnormally placed appendix New York J Med 50 2965 1950
- COKKINS A J Observations on mesenteric circulation J Anat 64 200 1930
- COLCOCK B P Intestinal obstruction due to gallstones Lahey Clin Bull 2 47 1940
- COLCOCK B P Treatment of acute obstruction of the small intestine S Clin North America 20 781 1940
- COLCOCK B P Internal herniation following sub total gastrectomy Lahey Clin Bull 8 233 1954
- COLE, W H Retroperitoneal hemorrhage simulating acute peritonitis JAMA 96 1472 1931
- COLE, W H Intestinal obstruction Canad MAJ 58 241 1948
- COLE W H Intestinal obstruction California Med 73 384 1950
- COLE, W H Intestinal obstruction Rocky Mountain M J, 47 667 1950
- COLE, W H Operability in the young and aged Ann Surg 138 145 1953
- COLEMAN E P MACUIRE, R H AND BENNETT D A Recurring jejunal intussusception Am J Surg 53 340 1941
- COLEY cited by THOMAS H O Contributions to Surgery and Medicine H K Lewis Co London 1883
- COLLER F A AND BUXTON R W Acute obstruction of the small bowel JAMA 140 135 1949
- COLLER F A CAMPBELL, K N VAUGHAN H H Job L V AND MOYER, C A Post operative salt intolerance Ann Surg 119 533 1944
- COLLER, F A AND MADDOCK W G Water and electrolyte balance Surg Gynec & Obst 70 340 1940
- CONNOLLY J E Hypertrophic pyloric stenosis in the adult Am J Surg 89 1123 1955
- CONOLE, F D Gallstone ileus New York J Med 50 1617 1950
- CONRAD H A AND ROBBINS F P Congenital atresia of the jejunum with meconium peritonitis J Internat Coll Surgeons 15 320 1951
- COOK J Ileocolic intussusception of traumatic origin Brit M J 1 526 1950
- COOKE, R F AND SEGAR, W E A proposed mechanism of extracellular regulation of muscle composition Yale J Biol & Med 25 83 1952
- COOPER, A Anatomy and Surgical Treatment of Crural and Umbilical Hernia p 87 Longman and Co London 1807
- COTTAM C I W AND COTTAM C Congenital atresia of the duodenum Journal Linct 62 83 1942

- COTT G AND IFRICH R Des sténoses intestinales tardives consecutives a l'etrangement d'hermia Iev gynec et chir alid 9 235 1905
- COURVOISIER L G *Caustisch statistische bestrage zur Pathologie und Chirurgie der Gallenwege* vol 12 F C W Vogel Co Leipzig 1890
- COWLEY L C AND HARRIS H N Perforation of the gallbladder a study of twenty five consecutive cases Surg Gynec & Obst 77 661 1943
- COX W Paralytic ileus J Kansas M Soc 40 12 1939
- CRAIG C Intestinal obstruction of small bowel M J Australia 2 146 1931
- CRAIG C Special methods in the diagnosis of subacute obstruction of the small intestine M J Australia 2 545 1934
- CRAIG R M HODGSON J R AND DOCKERTY M B Obstruction of the small intestine in infants and children a roentgenologic and pathologic study Am J Roentgenol 72 412 1954
- CRAWF A W Gallstone obstruction of the duodenum with sinus between gallbladder and duodenal bulb Am J Roentgenol 26 92 1931
- CREIGHTON R H AND JONES W R Enteric intussusception in an adult Minnesota Med 23 677 1940
- CRENSHAW J F Importance of fluoroscopic positioning of gastric tube JMA Alabama 20 208 1950
- CRICKLAIR, G J AND HIRATZKA H Intraoperative mercury granuloma Ann Surg 137 272 1953
- CRILE, G Some postoperative emergencies in gastrointestinal surgery S Clin North America 15 1007 1935
- CRILE G JR Medical aspects of intestinal obstruction M Clin North America 32 373 1948
- CROCE, E J AND WIPER T B Cecocolic intussusception in the adult Am J Surg 66 389 1944
- CROCKETT F B Review of literature on small bowel obstruction. Brit J Surg 37 314 1950
- CROMMELINCK G Volvulus intestinal guérison par le mercure métallique Ann Soc med d'Anvers Bruc 1840 also Arch med Belge 1 1840
- CROOKS Tr Med Chir Soc Edinburgh 2 38 1826
- CROSSIN H S AND CROSSIN D F *Foreign Bodies Left in the Abdomen* C V Mosby Co St Louis 1940
- CROWLEY R T Reflex changes in respiration induced by distention of the small intestine Arch Surg 44 707 1942
- CROWLEY R T AND JOHNSTON C G Therapeutic considerations in acute obstruction of the small intestine Internat Abstr Surg 73 1 1941
- CROWLEY R T AND WINFIELD J M Internal strangulating obstruction of the bowel Internat Abstr Surg 89 17 1949
- CROWLEY R T AND WINFIELD J M General factors governing the management of small bowel obstructions in children New York J Med 50 2943 1950
- CRUVEILHIER cited by CONNOLLY J E Hypertrophic pyloric stenosis in the adult Am J Surg 89 1123 1955
- CUBBINS W R AND FEY A Acute intestinal obstruction mechanical in type of the jejunum and ileum Illinois M J 65 203 1934
- CULLEN T S Intestinal obstruction due to a hole in the mesentery of the ascending colon JAMA 106 895 1936
- CULLIGAN J Intestinal obstruction due to calcified mesenteric glands Minnesota Med 21 482 1938
- CULLIGAN L Bowel obstruction occurring during postoperative period Minnesota Med 37 198 1954
- CUNNINGHAM D J *Textbook of Anatomy* 9th ed edited by J C BRASH Oxford University Press 1951
- CUNY J Rupture d'un aneurysme de l'aorte abdominale simulat un volvulus du colon pelvier Lyon chir 34 58 1937
- CURRY G J Gallstone intestinal obstruction case history J Michigan Soc 30 24 1931
- CURTIS H P AND NICHOI, W W Agnesia of the ileum and jejunum US Armed Forces M J 3 1707 1952
- CUTLER C W JR Acute intestinal obstruction in elderly patients Surg Gynec & Obst 94 481 1952
- CUTLER, E C, AND PIJOAN M Certain chemical factors in experimental high intestinal obstruction Surg Gynec & Obst 64 892 1937
- CUTLER, G D Mesenteric defect as a cause of intestinal obstruction Boston Med & Surg J 192 305 1925
- D'ABREU A J Unusual termination of intestinal obstruction due to a gallstone Brit M J 2 388 1945
- DANADE, R. AND DE GRAILLY Tubage duodénal rôle du facteur mécanique dans la sécrétion du suc duodénal Compt rend Soc de biol Paris 88 125 1923
- DANAVANT D AND WILSON H Intussusception of the appendix Ann Surg 135 287 1952
- DAVIS A Leukocytosis as an alarming sign in intestinal obstruction Acta chir belg 48 270 1949
- DANOWSKI T S WINKLER A W AND ELKINGTON J R The treatment of shock due to salt depletion comparison of the hemodynamic effects of isotonic saline of hypertonic saline and of isotonic glucose solution J Clin Invest 25 130 1946
- DARLING H C The treatment of intestinal obstruction in general practice M Press & Circular 190 430 1935
- DARNE, F Congenital atresia of small intestine Brit J Surg 34 315 1947
- DARROW D C The retention of electrolyte during recovery from severe dehydration due to diarrhea J Pediat 28 515 1946
- DARROW D C AND PRATT E L Fluid therapy relation to tissue composition and the expenditure of water and electrolyte JAMA 143 365 1950
- DAVID V C Chronic intestinal obstruction due to lesions of the large bowel Surg Gynec & Obst 64 281 1937
- DAVIS D L AND POYNTER C W M Congenital occlusions of the intestines Surg Gynec & Obst 34 35 1922
- DAVIS H A GASTER, J MARSH R L AND PRITEL, P A The effect of streptomycin in experimental

- strangulation of the bowel *Surg, Gynec & Obst* 67 63 1948
- DAVIS M B Intestinal obstruction from eating bran *JAMA* 97 24 1931
- DE GRAFF *Ciba Symposia* vol 5 February 1944
- DELPRAT G D AND WEEKS A Post operative intestinal obstruction *Am J Surg* 8 1189 1930
- DEMIKOVA P N Blood picture in acute ileus *Vestnik Khir* 56 356 1938 Abstr *JAMA* 112 1775 1939
- DENNEN E V AND BRODERICK T C Gallstone in intestinal obstruction *Ann Surg* 131 225 1950
- DENEVEY F Gastrointestinal obstruction simulating malignancy *Illinois M J* 66 336 1934
- DENNIS C Oblique aseptic end-to-end iliac anastomosis procedure of choice in strangulating small bowel obstruction *Surg Gynec & Obst* 77 225 1943
- DENVIS C Treatment of large bowel obstruction Transverse colostomy—incidence of incompetency of ileocecal valve experience at the University of Minnesota Hospital *Surgery* 15 713 1944
- DENNIS C AND BROWN S P Intestinal obstruction *Bull Hosp Univ Minn Staff Meet* 13 185 1942
- DENNIS C AND BROWN S P Treatment of small bowel obstruction *Surgery* 13 94 1943
- DENNIS C AND TOON R Bowel obstruction *Bull Hosp Univ Minn Staff Meet* 18 62 1946
- DE POTO A A Some observations and deductions of ileostomy in low acute mechanical obstruction a report of a series of cases *Am J Surg* 31 562 1936
- DEUTSCH I Intestinal obstruction in Hodgkin's disease *Rev Gastroenterology* 17 1134 1950
- DEVINE J A concept of paralytic ileus a clinical study *Brit J Surg* 34 158 1946
- DEVINE J W JR A new tube for use in treatment of intestinal obstruction *JAMA* 148 114 1952
- DEVINE, J W AND DEVINE J W JR Treatment of intestinal obstruction with the air vent tube *Virginia M Monthly* 79 17 1952
- DEVINE J W AND DEVINE, J W JR Duodenal intubation *Surgery* 33 513 1953
- DE WITT W F MORRISSEY P G JR AND FAILLA S D Obstruction of the small intestine from a primary calculus *Mil Surgeon* 92 34 1943
- DEYES E L AND MIDDLEMAN I C The treatment of fistula and obstruction of the small intestine by complete exclusion *Surg Gynec & Obst* 72 237 1941
- DICKENSON E H AND ZIMMERMAN L M Acute bowel obstruction secondary to regional enteritis *U S Naval M Bull* 44 835 1945
- DIEFFENBACH cited by TREVIS F *Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- DIEFNER Ileus stercoraceus durch anwendung des Mercurius vivus geheilt *Schweiz Ztschr Med Chir u Geburtsh* Zurich p 104 1847
- DITTEL, F C Importance for surgeon of author's method of intubation *Beitr klin Chir* 138 559 1926
- Discussion on the management of acute large bowel obstruction due to carcinoma *Proc Roy Soc Med* 44 779 1951
- DIXON C I Acute intestinal obstruction *New Orleans M & S J* 85 87 1932
- DIXON C F The medical and surgical management of the distended colon *Proc Inter state Post grad Med Assembly North America* Oct-Nov 1939
- DIXON C F AND BARCEY J A Volvulus of the sigmoid *West J Surg* 40 470 1932
- DIXON C F AND DEUTERMAN J L Acute intestinal obstruction *S Clin North America* 17 983 1937
- DIXON C F AND GREER R C Management of post operative intestinal obstruction complicated by hemorrhage on the basis of prothrombin deficiency *Minnesota Med* 23 169 1940
- DIXON C F AND MEYER A C Volvulus of the cecum *S Clin North America* 28 953 1948
- DIXON C F AND MILLER J M Volvulus of the cecum a postoperative complication *Minnesota Med* 21 250 1940
- DIXON C F AND WEISSMAN R F Management of intestinal obstructions *Arizona Med* 4 26 1947
- DOCK G The first aphorism of Hippocrates *Ann Int Med* 6 129 1932
- DODGEON H R, JR Geriatric surgery *Am J Surg* 79 417 1950
- DOERFLER Is the small intestine essential for life? *Zentralbl Chir* 1 1502 1923
- DONALDSON J K Intestinal obstruction *Arch Surg* 35 155 1937
- DONHAUSER J K AND KELLY E C Intussusception in the adult *Am J Surg* 29 673 1950
- DONOVAN R E AND GARAT J A Distension gaseosa en cirugia abdominal *Rev Asoc med argent* 59 149 1944
- DOTT N M Anomalies of intestinal rotation their embryology and surgical aspects with a report of 5 cases *Brit J Surg* 11 251 1923
- DOUGLAS J Recurring intestinal obstruction from gallstones *Ann Surg* 96 107 1932
- DOWD C N Resection of one third of the colon for irreducible intussusception in an infant five days old *Ann Surg* 57 713 1913
- DRASTEDT C A Some pharmacological considerations of intestinal obstruction *Illinois M J* 74 313 1938
- DRASTEDT C A LANG V G AND MILLET R F Relative effects of distention upon different portions of intestine *Arch Surg* 18 2257, 1929
- DRASTEDT C A AND MOORHEAD J J Immunity in intestinal obstruction *J Exper Med* 27 359 1918
- DRASTEDT I R Acute dilatation of the stomach *Text Practice of Surgery* chap 10 vol 6 W B Saunders Co Hagerstown Md 1955
- DRASTEDT L R Surgical treatment of acute intestinal obstruction *Surg Gynec & Obst* 50 191 1940

- DRAGSTEDT L I Some physiological principles involved in acute intestinal obstruction Illinois M J 74 320 1938
- DRAGSTEDT I K DRAGSTEDT C A MCCLINTOCK J R AND CHASE C S Intestinal obstruction A study of the factors involved in the production and absorption of toxic materials from the intestine J Exper Med 30 109 1919
- DRAGSTEDT I R MONTGOMERY M K FILLIS J C AND MATTHEWS W B Pathogenesis of acute dilatation of the stomach Surg Gynec & Obst 52 1075 1931
- DRAPER J W Experimental intestinal obstruction Report of further physiologic cytologic and chemical studies JAMA 57 1338 1911
- DRAPER J W AND JOHNSON R K The pathogenic omentum JAMA 88 3/6 1927
- DRAPER J W AND JOHNSON P K Chronic intestinal obstruction of the segmental type JAMA 94 683 1930
- DUFNAN F M VAL DEZ I C AND MURRAY J C Intestinal obstruction M Clin North America 14 1083 1931
- DUCKETT J W Intestinal obstruction in the newborn Ann Surg 116 321 1942
- DUFFY G S MISCOLL L AND MORSE S F Benign tumors of the stomach Rev Gastroenterol 10 31 1943
- DUJARDIN BEAUMETZ *Diseases of the Stomach and Intestines* Wm Wood and Co New York 1886
- DULAVANT D AND WILSON H Intussusception of the appendix Ann Surg 135 287 1952
- DUNCAN P A WEARN I S JACKSON H F AND WALDRON W S Successful surgical treatment of multiple atresias (aplasias) of the small intestine in a premature infant JAMA 123 764 1943
- DUNLOP J A Ogilvie's syndrome of false colonic obstruction a case with postmortem findings Brit M J 1 890 1949
- DURN D D AND SHEARBURN E W Jejunal intussusception Surgery 26 833 1949
- DUPUYTREN cited by TREVES F *Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- DU ROUX E Les torsions pathologiques du gros intestin et leur traitement chirurgical en deux parties Rev de gynec et de chir abd 19 319 1912
- DUAL R La rachianesthésie dans l'ileus aigu Resumes et conclusions de la discussion Bull et mem Soc nat de chir Paris 53 596 1927
- EASTON F R AND ADAMS J E Incipient volvulus of the caecum associated with left sided colon Surgery 1 920 1937
- EDFLMAN I S HALFY H B SCHLOERB P R SHELDON D B FRIIS HANSEN B J STOLL G AND MOORE F D Further observations on total body water 1 Normal values throughout the life span Surg Gynec & Obst 95 1 1952
- EDFLMAN I S JAMES A H AND MOORE F D Electrolyte composition of bone and the penetration of radiisodium and deuterium oxide into dog and human bone J Clin Invest 33 122 1954
- EDWARDS C I Acute intestinal obstruction due to mesenteric defects requiring massive resection JAMA 99 278 1932
- ELDER S A Primary malignant disease of duodenum Arch Surg 27 1087 1933
- ELIHOORN M A practical method of obtaining the duodenal contents in man M Rec 77 98 1910
- ELIHOORN M Historical sketch of duodenal tube Am J M Sc 151 202 1916
- ELIHOORN M New tip for gastroduodenal tubes JAMA 86 1615 1926
- ELIHOORN M Nasal simultaneous gastroduodenal aspirator Surg Gynec & Obst 72 48 1941
- ELIHOORN M Weighted nasal gastroduodenal tube Am J Surg 52 518 1941
- ELIASON E L AND JOHNSON J Diagnostic features of ileus Radiology 26 342 1936
- ELIASON F L AND WELTY R F A ten year survey of intestinal obstruction Ann Surg 125 57 1947
- ELKAN W E Acute mesenteric vascular occlusion following mumps J Internat Coll Surgeons 20 259 1953
- ELKINGTON J R DANOSKI T S AND WINKLER A W Hemodynamic changes in salt depletion and in dehydration J Clin Invest 25 120 1946
- ELKINGTON J R WINKLER A W AND DANOSKI T S The importance of volume and tonicity of the body fluids in salt depletion shock J Clin Invest 26 1002 1947
- ELKINGTON J R WINKLER A W AND DANOSKI T S Transfers of cell sodium and potassium in experimental and clinical conditions J Clin Invest 27 74 1948
- ELLIS J W The cause of death in high intestinal obstruction Ann Surg 75 429 1922
- ELMAN R The treatment of late acute intestinal obstruction Surg Gynec & Obst 56 175 1933
- ELMAN R The danger of sudden deflation of acutely distended bowel in late low intestinal obstruction Am J Surg 26 438 1934
- ELMAN R *Parenteral Alimentation in Surgery* Paul B Hoeber Inc New York 1947
- ELMAN R AND COLE W H Pathological changes in the liver accompanying obstruction and strangulation Proc Soc Exper Biol & Med 29 1274 1932
- ELMAN R AND HARTMAN A F Experimental obstruction of the terminal duodenum and ileum Surg Gynec & Obst 53 307 1931
- ELMAN R LEMMER R A WEICHELBAUM T E OWENS J G AND YORE R W Minimum post operative maintenance requirements for parenteral water sodium potassium chloride and glucose Ann Surg 130 703 1949
- ELMAN R AND MCCAUGHAN J M On collection of entire external secretion of pancreas under sterile conditions and fatal effect of total loss of pancreatic juice J Exper Med 45 561 1917

- ELSVEN H I The vagaries and wanderings of gall stones *M News* 72 164 1898
- ELSON K A AND DROSSNER J L Intubation studies of human small intestine effect of atropine and belladonna on motor activity of small intestine and colon *Am J Digest Dis* 6 589 1939
- EMHARDT J W The management of acute intestinal obstruction *J Indiana M A* 23 356 1930
- The enema *Cila Symposia* vol 5 February 1944
- FRASISTRATUS cited by MASSENGILL S F *A Sketch of Medicine and Pharmacy* S E Massengill Co Bristol Tenn 1940
- ERICSEN O C AND GREENFIELD R F Volvulus of the ascending colon with report of a case *JAMA* 133 616 1947
- ERNBERGER G H Acute intestinal obstruction due to fetal peritonitis *Am J Surg* 18 322 1932
- ERNST N P A case of congenital atresia of the duodenum treated successfully by operation *Brit M J* 1 644 1916
- ESSEN MOELLER Ueber Ileus in der Schwangerschaft und bei Entbindung *Zentralbl Gynak* 40 1014 1916
- ESSENSEON L AND GINZBURG L Volvulus of the sigmoid *Am J Surg* 77 240 1949
- FESTES W L Jr Enteritis of the obstructed loop following entero anastomosis for intestinal obstruction *Ann Surg* 105 871 1937
- ESTES W L AND HOLM C E Fate of obstructed loop in intestinal obstruction following anastomosis around obstruction without resection *Ann Surg* 96 924 1932
- EVANS C H Atresias of the gastrointestinal tract *Internat Abstr Surg* 92 1 1951
- EVANS E I The mechanism of shock in intestinal strangulation *Ann Surg* 117 28 1943
- EVANS E I AND BIGGER I A Early recognition and management of intestinal strangulation *JAMA* 133 513 1947
- EVANS H M AND BRENER A G Resection of the small intestine in the dog *Johns Hopkins Bull* 18 477 1907
- EVANS W A Jr Obstructions of the alimentary tract in infancy *Radiology* 51 23 1948
- EVANS W A Jr Intussusception (editorial) *Am J Roentgenol* 61 851 1949
- EVANS W REYNOLDS L JARRE H MELLINS H AND FIGIEL L Personal communication to authors as to competence of ileocecal valve in routine barium enema
- EVERT J A Primary nonspecific ulcers of the small intestine *Surgery* 23 185 1948
- EWING J *Neoplastic Diseases A Treatise on Tumors* 4th ed p 602 W B Saunders Co Philadelphia 1940
- FABER A *Klin Wchnschr* 34 643 1897 cited by SPONG D H JR POLLACK W F AND MACK M A The anemias of intestinal stagnation *Western J Surg* 61 217 1953
- FABRICIUS HILDANUS cited by TREVES F *Intestinal Obstruction Parishes and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- FAGGE C H On intestinal obstruction *Guy's Hosp Rep* 14 272 1868
- FAGGE C H AND HOWES H G A case of intussusception in an adult without symptoms of strangulation treated successfully by abdominal section *Med Chir Tr* 59 85 1876
- FAIR G L Foreign body in the abdomen causing obstruction *Am J Surg* 86 472 1953
- FAIRCHILD F R The mechanical factors relating to obstruction following gastroenterostomy *Tr West S A* 39 93 1929
- FAIRCHILD F R Obstruction following gastroenterostomy its mechanical factors *Calif & West Med* 33 1, 1930
- FAIRCHILD F R Age and operations *S Clin North America* 13 33 1933
- FALCONER M A AND LYALL A Blood chemistry in intestinal obstruction changes in response to treatment *Lancet* 2 1472 1937
- FALCONER M A OSTERBERG A F AND BARGEN J A Intestinal obstruction in man Alterations in the serum bases and their significance *Arch Surg* 38 869 1939
- FALLIS L S AND BARRON J Gastric and jejunal alimentation with fine polyethylene tubes *Arch Surg* 65 373 1952
- FALLIS L S AND WARREN K W Irreducible intussusception in infants *Surg Gynec & Obst* 81 384 1945
- FANG H C AND LOUCKS H H Decompression of the gastro intestinal tract application of principles of mechanical decompression to treatment of various surgical conditions of abdomen *Chinese M J* 50 201 1936
- FANTUS B AND KOPSTEIN G Does bran produce intestinal obstruction? *Am J Digest Dis* 7 60 1940
- FARBER S Congenital atresia of the alimentary tract *JAMA* 100 1753 1933
- FARRAR T Duodenal obstruction in the newborn *Memphis M J* 24 199 1949
- FEDERSCHMIDT cited by CUTLER G D AND SCOTT H W Transmesenteric hernia *Surg Gynec & Obst* 79 309 1944
- FEGGOTTER S Congenital duodenal atresia *Lancet* 2 188 1940
- FENN W O The role of potassium in physiological processes *Physiol Rev* 20 377 1940
- FERSTEN M Intestinal obstruction during pregnancy *New England J Med* 242 977 1950
- FERGUSON I T Postoperative intestinal obstruction *South Med J Surg* 96 217 1954
- FIFE A AND CUBBINS W R Acute mechanical intestinal obstruction mortality with and without enterotomies *Surg, Gynec & Obst* 60 738 1935
- FICARRA B J Cecostomy in large bowel obstruction associated with mega cecum *New York J Med* 49 2191 1949
- FIELDS A Physiotherapy in ancient Chinese medicine *Am J Surg* 79 613 1950

- FICI, F. A. The etiology and treatment of cicatricial stenosis of the larynx and trachea. *South M J* 40 14 1947
- FICHEL, L. S. AND FICHEL, S. J. Volvulus of the cecum and ascending colon. *Radiology* 61 496 1953
- FICHEL, I. S. AND FICHEL, S. J. Studies on intestinal obstruction. *Crace Hosp Bull*, 32 65 1954
- FICHEL, L. S. AND FICHEL, S. J. Studies on intestinal obstruction. *Crace Hosp Bull* 32 17 1955
- FICHIOLINI, F. AND CINTRA DA PRADO, F. Causes of failure in intubation. *Brasil med* 45 341 1931
- FILHO, S. How to pass pyloric sphincter quickly during intubation. *Acta med Plo de Janeiro* 3 30 1939
- FINE, J. Mechanical obstruction of the small intestine. *New England J Med* 219 223 1938
- FINE, J., BANKS, B. M., SEARS, J. B. AND HERMANSON, I. Treatment of gaseous distention of intestine by inhalation of 95% oxygen: description of apparatus for chemical administration of high oxygen mixtures. *Ann Surg* 103 375 1936
- FINE, J., FLICHS, I. AND GENDEL, S. Changes in plasma volume due to decompression of distended small intestine. *Arch Surg* 40 710 1940
- FINE, J., POSENFELD, L. AND GENDEL, S. The role of the nervous system in acute intestinal obstruction. *Ann Surg* 110 411 1939
- FINNEY, J. M. T. Acute intestinal obstruction. *Surg Gynec. & Obst* 32 402 1925
- FISHER, W. Foreign bodies arrested in the duodenum. *Ann Surg* 70 479 1919
- FITZWILLIAMS, D. C. L. Pathology and etiology of intussusception from study of 1000 cases. *Lancet* 1 628 1908
- FLEMING, A. On a remarkable bacteriolytic element found in tissues and secretions. *Proc Roy Soc London*, 93 306 1927
- FLEMMING, C. Retrograde intussusception. *Lancet* 2 1136 1937
- FLINT, J. M. The effect of extensive resections of the small intestine. *Johns Hopkins Bull* 23 127 1912
- FLYNN, J. M. Intestinal obstruction at terminal ileum caused by large irregular gallstone. *Am J Roentgenol* 44 69 1940
- FOERSTER, cited by CHERRY, J. W. AND HILL, L. R. Leiomyoma of the jejunum. *Arch Surg* 62 580 1951
- FOISIE, P. S. Intestinal obstruction following abdominal battle wounds. *New England J Med* 234 498 1946
- FOLLEY, J. H. The medical management of intestinal obstruction. *New England J Med* 278 606 1943
- FORESTER, V. R. Gallstone ileus. *Gastroenterology* 15 679 1950
- FORSALL, T. Intussusception of the vermiform appendix. *Brit J Surg* 40 305 1953
- FORSTER, A. C. Production of hyper and hypomotility of musculature of small bowel in human experimental intubation: normal peristaltic activity: effect of mechanical stimulation. *Ann Surg*, 112 340 1940
- FORTY, F. Enteric intussusception treated by resection. *Lancet* 1 72 1940
- FOSS, H. L. AND SUMMERS, J. D. Intestinal obstruction from gallstones. *Ann Surg* 115 721 1942
- FOSTER, W. O. AND HAUSLER, R. W. Studies of acute intestinal obstruction: acute strangulation. *Arch Int. Med* 34 697 1924
- FOX, C. L. JR., LASKER, S. E., WINFIELD, J. M., AND MERSHEIMER, W. L. Albumen potassium sodium and chloride redistribution and erythrocyte loss after surgical trauma and extensive burns. *Ann Surg*, 140 254 1954
- FOX, A. I., MANTEL, F. J. AND RABENS, J. T. Acute toxic nephritis complicating acute obstruction of the small intestine. *J A.M.A.*, 96 943 1931
- FOX, P. F. AND BRENNAN, J. E. Ruptured omphalocele and jejunal atresia. *Ann Surg* 133 123 1951
- FOX, P. F., AND CRAWFORD, O. W. Duodenal obstruction situs inversus and rotation of the colon. *Surgery* 27 896 1950
- FRAENKEL, G. J. Carcinoid causing obstruction. *Lancet* 1 404 1948
- FRANCESCO, M. Nota clinica su di un raro incidente nella corsa del sondaggio duodenale. *Gasetta d'osp* 54 1050 1933
- FRANKAU, C. Prognosis in acute intestinal strangulation. *Lancet* 1 103 1935
- FRANZAS, F. Nachuntersuchung von Patienten mit Spliserohren und Magenverengungen in Finnland. *Acta chir scandinav* 89 1 1943
- FRASER, F. C. Recurrent volvulus of the sigmoid colon cured by complete sigmoidectomy. *Indian M Gaz* 68 519 1933
- FREDET, P. La stenose du pylore par hypertrophie musculaire chez les nourrissons. *Bull et mem Soc d'chirurgiens de Paris* 47 451 1921
- FREDERICK, R. Acute intestinal obstruction. *J Nat M. A.*, 27 68 1935
- FREEMAN, S. Parenteral administration of fats. *Bull Northwest Univ M School* 28 113 1954
- FREED, W. I., ZIMMEL, A., STROUD, C. S. AND FRIES, J. W. Fecaloma in the sigmoid colon. *Gastroenterology* 29 446 1955
- FRIEDEL, M. T., AND WAKEFIELD, E. G. The ileocecal valve in man. *Mayo Clin. Proc Staff Meet* 16 705 1941
- FRIES, J. A. Intestinal gases of man. *Am J Physiol.*, 16 468 1906
- FRIMANN DAHL, J. Postoperative Röntgenuntersuchungen Diaphragmabewegungen und der postoperative Venenstrom postoperative Lungenembolien. *Acta chir scandinav*, 76 1 1935
- FRIMANN DAHL, J. Roentgenological examinations of ileus. *Acta radiol* 28 330 1947
- FROMME, A. Rare type of ileus after gastroenterostomy. *Zentralbl. Chir* 47 1505 1920
- FÜTH, H. AND OPIESEN, Weiterer Beitrag zur Frage der Verlagerung der Zykum in der Schwangerschaft. *Dtsch. h. m. l. Wchnschr* 54 812 1928

- FURNISS H D Modified Wangenstein suction drainage Surg Gynec & Obst 68 118 1939
- GABRIEL L T CAMPBELL D A AND MUSSELMAN M M Volvulus of the sigmoid colon Gastroenterology 24 378 1953
- GAERTNER W Atresia and stenosis of the intestine in association with ileus in neonates Bruns Beitr klin Chir 186 21 1953
- GAGE H C Discussion Proc Roy Soc Med 30 33 1937
- GAGE M AND HOSOI K Histological changes observed in the intestinal wall following simple mechanical obstruction in rabbits Proc Soc Exper Biol & Med 32 1651 1935
- GALEN *De Alimentoium Facultatibus* vol 3 Calveum Paris 1530
- GALLART M F Physiopathology of the syndrome of acute obstruction of the small intestine El Dia Medico 17 317 1945
- GALLISON D T Gastro intestinal allergy as a cause of intestinal obstruction New England J Med 211 164 1934
- GAMBLE H A The role of obstructive enterostomy in the treatment of obstruction of the small intestine South M J 42 490 1949
- GAMBLE J L *Chemical Anatomy Physiology and Pathology of Extracellular Fluid* Harvard University Press Cambridge Mass 1947
- GANN M E AND HOFFMAN E Congenital atresia of the sigmoid Am J Surg 83 605 1952
- GARDNER C E JR The surgical significance of anomalies of intestinal rotation Ann Surg 131 879 1950
- GARDNER C I JR AND HART D Anomalies of intestinal rotation as a cause of intestinal obstruction report of 2 personal observations review of 103 reported cases Arch Surg 29 942 1934
- GARDNER F T Bell's toll for primis Surg Gynec & Obst 86 377 1948
- GARRELY L J AND ATCHIN R M Intussusception J Michigan M Soc 43 665 1944
- GASTON E A AND COSTIN M E Closed loop obstruction of the ascending colon with incomplete descent of the cecum simulating acute cholecystitis New England J Med 239 705 1948
- GATCH W D Intestinal obstruction *Lectures in Practice of Surgery* vol 7 chap 7 pp 1-48 W F Prior Co Hagerstown Md 1955
- GATCH W D The blood chemistry toxemia and mechanics of advanced intestinal obstruction with deduction on treatment Illinois M J 60 236 1931
- GATCH W D AND BATTERSBY J S The two stages of bowel distention Arch Surg 44 108 1942
- GATCH W D TRUSLER H M AND AYERS K D Causes of death in acute intestinal obstruction Surg Gynec & Obst 46 332 1938
- GATTELLER J MEYER I AND ICHER I Lesions du cecum Arch mal app digest 21 20 1931
- GATLING R R AND KIRBY SMITH H T Volvulus of the sigmoid colon Ann Surg 128 1073 1948
- GALDINO M AND LEVITT M I Influence of the adrenal cortex on body water distribution and renal function J Clin Invest 282 1487 1949
- GAY B B JR LEITCH T F AND RIGERS J A Meckel's diverticulum as a cause of intussusception J M A Georgia 42 15 1953
- GENDEL S AND LINE J The effect of acute intestinal obstruction on the blood and plasma volume Ann Surg 110 25 1939
- GERARD R W Chemical studies on intestinal intoxication presence and significance of histamine in an obstructed bowel J Biol Chem 52 111 1922
- GERWIG W H JR Volvulus of the colon Arch Surg 60 721 1950
- GERWIG W H JR AND STONE H B Intercutaneous intussusception in adults Surg Gynec & Obst 76 95 1943
- GIBSON C L A study of 1000 operations for acute intestinal obstruction and gangrenous hernia Ann Surg 32 486 1900
- GIBSON C L Ball valve tumor of the stomach Ann Surg 45 130 1907
- GIBSON R H DOCKERTY M B AND DIXON C F Intussusception in infants and children S Clin North America 29 1141 1949
- GILDER H REDO S F BARR D AND CHILD C G III Water distribution in normal subjects and in patients with Laennec's cirrhosis J Clin Invest 33 555 1954
- GILSON O AND GRATIS A Le phenomene d'Arthur au catgut cause insoupconnee d'adherence post-operatoire J chir 51 43 1938
- GINZBURG L X ray diagnosis of acute intestinal obstruction without use of contrast media Ann Surg 96 368 1932
- GISSANE W Recurrent intussusception of the jejunum Brit J Surg 25 608 1938
- GIUS J A AND PETERSON C G Postoperative ileus and related gastrointestinal complications Internat Abstr Surg 79 265 1944
- GLASS B A AND ABRAMSON P D Volvulus of cecum due to lithopedion Am J Surg 86 348 1953
- GLAZER I AND ADLERSBERG D Volvulus of the colon a complication of sprue Gastroenterology 24 159 1953
- CLEVELAND F A De l'enteropse Irc se med belge 41 57 1889
- CLEVEN J P The treatment of peritonitis from the standpoint of intestinal obstruction Kentucky M J 25 305 1937
- GLENN I M AND LAUCKNER I L Intestinal intubation in gynecologic intestinal complications Am J Obst & Gynec 39 836 1940
- GLICK D L AND ANSTINE W Intubation of the line es intestinales J Pharmacol & Exper Therap 65 381 1951

- COVER D M AND HAMANN C A Intestinal obstruction in the newborn due to congenital anomalies Ohio M J 36 833 1940
- GLORY D M SMITH S AND FITZES O Multiple atresia of the small intestine Ann Surg 116 337 1942
- CONERY N C Volvulus of caecum with reversed rotation of mid gut Brit M J 1 911 1945
- COFFLER C A Abortion humani mostrosi Hist Anatom Misc Acad Nat Curios 1683 Norimb 1684 decuria 2 11 311
- GOLDBERG H A case of intestinal obstruction in newborn Med Klin 22 1801 1926
- GREEN I Abnormalities of small intestine in nutritional disturbance Radiology 36 207 1941
- GREEN I Case of intension of the small intestine Arizona Med 11 39 1954
- GOLLEN I FEICH C C JR AND SWENSON I C The roentgen ray examination with the Miller Abdominal tube Radiology 35 521 1940
- GOLDEN T AND STOUT A P Smooth muscle tumors of the gastrointestinal tract and retro peritoneal tissues Surg Gynec & Obst 73 784 1941
- GOLDMAN I Intestinal strangulating obstruction with negative roentgenologic findings Surgery 13 834 1943
- GOLDSTEIN M S DYER C L AND ZIFFREN S F Intestinal obstruction in the aged J Am Geriatrics Soc. 1 205 1953
- GUTHRIE J C LLOYD-DAVIS O L AND ROBERTSON I C Small gut obstruction following combined excision of the rectum with special reference to strangulation around the colostomy Brit J Surg. 38 467 1951
- GOLUB M The advisability of immediate colonic irrigation following barium enema Radiology 22 486 1934
- GOODALL H W cited by KIRKMAN N F BARLOW T AND MOTTISHIELD S A case of persistent intussusception in an adult Brit J Surg 28 476 1941
- GOOD T V AND NEWBURN W R Intestinal obstruction associated with defects in the broad ligaments of the uterus Am J Surg 65 127 1944
- GOODYEAR F S Retrograde intussusception of cecum into ileum New York J Med 38 1397 1938
- GORDINIER H C AND SAMPSON J A Diverticulitis (not Meckel's) causing intestinal obstruction multiple mesenteric (acquired) diverticula of the small intestine JAMA 46 1585 1906
- GORELIK A N Prognosis of coronary and rheumatic patients after cardiopericardiomyectomy operation Tr Amer Coll of Cardiology 4 148 1955
- CORMAN C F Intestinal obstruction New Orleans M & S J 89 249 1936
- GOULD G M AND PYLE W L Anomalies and Curiosities in Medicine Sydenham Publishers New York 1937
- GRACE W J AND BARR D P Complication of alkalosis Am J Med 4 331 1948
- GRAHAM ROBERT L et al communication authors GRAHAM ROBERT L A case of intestinal obstruction Brit M J 1 645 1935
- GRANET L Sigmoid rectal intussusception and the unstable colon Am J Digest Dis 19 32 1952
- CRANFYT F AND PEARL S S Sigmoid rectal intussusception with incarceration Am Pract Digest Treat 3 365 1952
- GRAVELLE I J AND GREEN R L Lymphangiomas of the jejunum Grace Hosp Bull 30 37 1952
- GRAVES cited by DRAGSTED C A Some pharmacological considerations of intestinal obstruction Illinois M J 74 313 1938
- GRAVES G Y Acute mechanical intestinal obstruction Kentucky M J 32 193 1934
- GREEN M T Surgical significance of derangement of intestinal rotation and distribution Surg Gynec & Obst 53 734 1931
- GREENE I I AND GREENE J M The early diagnosis of acute intestinal obstruction Illinois M J 61 506 1932
- GREENLEE R M AND BERRY W H Intestinal obstruction due to gallstones New York J Med 53 1342 1953
- GREENWOOD W F HAIST R F AND TAYLOR A B The plasma potassium following intestinal obstruction in dogs Surgery 7 280 1940
- GREGG H A AND SMITH F G Radiographic demonstration of localized mesenteric thrombosis Radiology 61 379 1953
- GREGORY L J PRIEST R S AND BARRON J Cholecystoduodenocolic ligament causing high grade obstruction of the duodenum Gastroenterology 23 659 1953
- GRIEVE S Paralytic ileus due to potassium depletion South African M J 27 153 1953
- GRIFFIN W D BARTON G R AND MEYER K A Volvulus of the sigmoid colon Surg Gynec & Obst 81 287 1945
- GRIMSON K S AND HODGE G B Prolonged intubation suction and deferred or delayed surgery in treatment of multiple adhesive obstructions of the small intestine Surg Gynec & Obst 78 316 1944
- GRINNELL R S Distal intramural spread of carcinoma of the rectum and recto sigmoid Surg Gynec & Obst 99 421 1954
- GROFFER M J Retrograde enteric intussusception Ann Surg 112 344 1940
- GROSS cited by GOULD G M AND PYLE W L Anomalies and Curiosities of Medicine Sydenham Publishers New York 1937
- GROSS M A duodenal tube preliminary communication New York M J 91 77 1910
- CRON R E Surgery of Infancy and Childhood W B Saunders Co Philadelphia 1953
- GROSS I F AND CHRISTENSEN T C Annular pancreas producing duodenal obstruction Ann Surg 119 759 1944
- CRON R F AND WARF P F Intussusception in children New England J Med 239 645 1948
- CRUTHICK F The axial rotation of colon through so-called physiological volvulus Acta radiol 15 153 1934

- GRUBER G B Survey of periarteritis nodosa *Klin Wchnschr* 4 1972 1925
- GRULLE cited by ERNSBERGER G H Acute intestinal obstruction due to fetal peritonitis *Am J Surg* 18 322 1932
- GRUTZNER cited by HOWELL W H *A Text Book of Physiology* 9th ed W B Saunders Co Philadelphia 1925
- GUIBAL J AND CUENOT A Importance of blood as a factor in peritoneal irritation *Presse med* 41 582 1933
- GUIGNARD P E *De retrecissement et de l'obliteration de l'intestin dans les hernies* Paris 1846
- GUTHRIE D Post operative ileus its early recognition and control *New York J Med* 31 1021 1931
- GUTHRIE D AND NILES J S Elective surgery in the aged *S Clin North America* 28 1341 1948
- GUTMANN J H Ileus due to migrating gallstone *Am J Surg* 30 548 1935
- GUZMAN I C AND PASQUAL A A Mechanical intestinal obstruction *Philippine J Surg* 6 170 1951
- HAAG H B AND TALIAFERRO I Effect of ascorbic acid on guinea pig colon *Proc Soc Exper Biol & Med* 45 479 1940
- HADEN R L AND ORR T G Chemical changes in the blood of man after acute intestinal obstruction An indication for treatment with sodium chloride *Surg Gynec & Obst* 37 465 1923
- HAEREM S DACK G M AND DRAGSTEDT L R Acute intestinal obstruction I The role of bacteria in closed jejunal loops II The permeability of obstructed bowel segments of dogs to *Clostridium botulinum* toxin *Surgery* 3 339 1938
- HAGGARD W D AND FLOYED W O Repeated resections for intussusception due to familial tumors of the small intestine *Am J Surg* 28 428 1935
- HAIGHT C Congenital atresia of esophagus with tracheo-esophageal fistula *Ann Surg* 120 623 1944
- HALL M R Roentgenological diagnosis of volvulus of sigmoid megacolon with report of two cases *Am J Roentgenol* 39 925 1938
- HALLER A VON *Elementa Physiologiae Corporis Humani* 8 v Lausanne 1757-1758
- HALSTEAD A E Intestinal obstruction from Meckel's diverticulum *Ann Surg* 35 471 1902
- HANAKER W D A unique case of bowel obstruction *JAMA* 62 204 1914
- HAMILTON A T Volvulus of the cecum *South M J* 42 138 1949
- HAMILTON J F Pre operative preparation and post operative care of the aged *J Tennessee M A* 41 336 1948
- HAMILIN E JR Non operative reduction of acute volvulus of the sigmoid *New England J Med* 247 835 1952
- HAMMAN J AND ALLDIS D Acute intestinal obstruction *South African J Clin Sc* 1 73 1950
- HAND B H AND CHURCH W F Gallstone ileus recurrence in the cat *Am J Surg* 59 72 1943
- HANDLEY R S Chronic ileus caused by malignant invasion of the posterior abdominal wall *Brit M J* 1 891 1949
- HANDLEY W S Paralytic ileus in acute appendicitis *Proc Roy Soc Med* 29 1 1935
- HARDA J D AND DRABKIN D L Measurement of body water techniques and practical application *JAMA* 149 1113 1952
- HARGER J R AND WILKEY J L Management of post operative distention and ileus *JAMA* 110 1165 1938
- HARKINS H N Intussusception due to invaginated Meckel's diverticulum *Ann Surg* 98 1070 1933
- HARNISTON G J AND CRAGUN W F Acute intussusception due to torsion of Meckel's diverticulum *Rocky Mountain M J* 46 291 1949
- HARPER W H AND BLAIN A III The effect of penicillin in experimental intestinal obstruction *Bull Johns Hopkins Hosp* 76 221 1945
- HARRINGTON S W Diaphragmatic hernia of children *Ann Surg* 115 705 1942
- HARRIS I I Intestinal intubation in bowel obstruction *Surg Gynec & Obst* 81 671 1945
- HARRIS F I AND GORDON M Intestinal intubation in small bowel distention and obstruction *Surg Gynec & Obst* 86 647 1948
- HARRIS M L Constrictions of the duodenum due to abnormal folds of the anterior meso-gastrum *JAMA* 62 1211 1914
- HARROP G A NICKOLSON W M SOFFER L J AND STRAUSS M Extracellular and intracellular water loss during supranal insufficiency in the dog *Proc Soc Exper Biol & Med* 32 1312 1935
- HARTMANN A F AND ELMAN F Effects of loss of gastric and pancreatic secretions and methods for restoration of normal conditions in body *J Exper Med* 50 387 1929
- HARTMANN A F AND SMYTH F S Chemical changes in body occurring as a result of vomiting *Am J Dis Child* 32 1 1926
- HARTMANN H AND PILLIET A H Note sur une variete de typhlite tuberculeuse simulant les cancers de la region *Bull Soc anat de Paris* 66 471 1891
- HARTWELL J A AND HOGLET J P Experimental intestinal obstruction in dogs with especial reference to cause of death and treatment by large amounts of normal saline solution *JAMA* 59 82 1912
- HARVEY S cited by CUITION Medical and surgical treatment of gall stone obstructing the intestines *Lancet* 1 124 1888
- HARVEY S C Congenital variation in the peritoneal relations of the ascending colon cecum appendix and terminal ileum *Ann Surg* 67 641 1918
- HATHFIELD C A BUYERS R A AND WALKING A A Fluorescein—its use in determining the viability of strangulated intestine *Surg Gynec & Obst* 81 530 1945

- HATFIELD M. Intrinsic antenatal intestinal obstruction at the ileum with peritonitis. Kentucky M J 37 481 1939
- HAYES W I. Intestinal obstruction caused by colloidal aluminum hydroxide. JAMA 113 1564 1939
- HAWTHORNE H K AND NEHR I JR. The management of intestinal obstruction. M Clin North America 33 1455 1949
- HAYES M A. Water soluble vitamin requirements in surgical convalescence. Ann Surg 140 661 1954
- HAYD J P. Medical allusion in Don Quixote. Ann med History 6 169 1934
- HIBBERG E A. Intubation ileus. Minnesota Med 23 94 1940
- HEIDENHAIN I. Arch klin Chir 55 211 1897
- HELLMER H. Intussusception in children: diagnosis and therapy with barium enema. Acta radiol 65 1 1948
- HELLFENS A V AND NEMMI P. Spontaneous obstruction of gastroenterostomy and complete detachment of intestinal loop. Ann chir et gynae Fenniae 40 121 1951
- HELMS J S JR. Intestinal obstruction in infants due to malrotation of the bowel associated with volvulus. Am J Surg 79 603 1950
- HEMMETTER J C. Gastric lavage with a continuous current: an improved recurrent stomach tube with history and technique of the subject. New York M J 62 819 1895
- HEMMETTER J C. History of discovery of duodenal intubation. M Pec 99 341 1921
- HENDERSON I I AND BALFOUR D C. Benign tumors of the stomach. Ann Surg 85 354 1977
- HENDON C A. The treatment of intestinal obstruction. Kentucky M J 31 65 1933
- HENDON G A. The treatment of postoperative ileus. Kentucky M J 31 204 1933
- HERODOTUS. Histories trans by A D GODLEY Loeb Classical Library. New York 1936
- HERRIN R C AND MEER W J. Distention as a factor in intestinal obstruction. Arch Int Med 51 152 1933
- HERMANN F T. Some great Arabians. Minnesota Med 12 214 1929
- HERMANN S F AND HIGGINS G M. Intestinal permeability in obstruction of the colon: an experimental study. Am J M Sc 179 365 1930
- HERSHEY C G. Treatment of small bowel obstruction and ileus by intestinal intubation. Proc Staff Conf Wheeling Clinic 10 101 1940
- HERTZ J. Duodenal obstruction. J Internat Coll Surgeons 13 729 1950
- HESCHL cited by RAVID J M. In discussion of paper by Wakefield and Friedell. JAMA 116 1889 1941
- HESS A F. The use of a simple duodenal catheter in the diagnosis and treatment of certain cases of vomiting in infants. Am J Dis Child 3 133 1912
- HUBLEIN G W THOMPSON W D AND SCULLY J P. Effect of vitamin B complex deficiency on gastric emptying and small intestinal motility. Am J Roentgenol 46 866 1941
- HUFER C J AND ANDREWS W D. The effect of adrenal cortical extract in controlling shock following the injection of aqueous extracts of closed intestinal loops. Ann Surg 100 734 1934
- HUBBARD J S. Treatment of intestinal obstruction in the field and aboard ship. US Naval M Bull 44 87 1945
- HILL F C O LOUCHLIN B J AND STONER M. Peritoneal aspiration in the diagnosis of strangulated bowel. Surg Gynec & Obst 74 121 1942
- HILTON H D AND WALGH J M. Volvulus of the sigmoid colon. Arch Surg 62 437 1951
- HITCHIEY P R. Recurrent gallstone ileus. New England J Med 223 174 1940
- HITCHIEY P R. Gallstone ileus. Arch Surg 46 9 1943
- HINDMARSH T A STEWART A W AND MORRISON B. The Mikulicz resection operation for gangrenous intussusception in infants. Brit M J 2 382 1943
- HINSON A. Failures in the use of Miller Abbott tube in intestinal obstruction. J South Carolina M A 38 284 1942
- HINTON D AND STEFFNER C A. Recurrent volvulus of the sigmoid colon. Ann Surg 116 147 1942
- HIPPOCRATES translated by LITTRE E. Œuvres Complètes d'Hippocrate 10 v J B Bailliere Paris 1839-1861
- HIPPLEY P L. Intussusception and its treatment by hydrostatic pressure: based on an analysis of one hundred consecutive cases so treated. M J Australia 2 201 1926
- HIRSCHSPRUNG H. Et Tilfælde af subakut Tarmnævnation. Hospitals tid 3 321 1876
- HIRSCHSPRUNG H. 107 Fälle von Darmnævnation bei Kindern behandelt im Königin Louise Kinderhospital in Kopenhagen während der Jahre 1871-1904. Mitt a d Grenzgeb d Med u Chir 14 555 1905
- HOAG C L AND SAUNDERS J B DE C M. Jejunoplasty. Surg Gynec & Obst 62 702 1939
- HOBBSON A. Volvulus of the small intestine. Brit M J 2 399 1950
- HOBSON C J. A case of intestinal obstruction due to gallstones. Brit J Radiol 16 185 1943
- HOFFMANN A. Ileus bei vorderer Gastroenterostomie. Zentralbl Chir 67 691 1937
- HOFFMAN I L. Spontaneous evacuation of metallic mercury from vermiform appendix. Bull US Army M Dept 8P802 1948
- HOFFMAN W H AND PACK G T. Cancer of the duodenum. Arch Surg 35 11 1937
- HOGUET J B AND HARTWELL J A. Experimental intestinal obstruction in dogs with especial reference to cause of death and the treatment by large amounts of saline solution. JAMA 59 82 1912
- HOLDEN W B. Surgical treatment of acute intestinal obstruction. Surg Gynec & Obst 50 184 1930
- HOLINGER P H AND LOEB W J. Feeding tube stenosis of larynx. Surg Gynec & Obst 83 253 1946

- HOLT R J Pathology of acute strangulation of the intestine Brit J Surg 21 383 1934
- HONOR W H AND SMATHERS N M A double lumened plastic tube for intestinal intubation Arch Surg 55 498 1947
- HOTZ G Beitrage zur Pathologie der Darmbewegungen Mitt a d Grenzgeb d Med u Chir Jena 20 257 1909
- HOWARD N J cited by DUNN D D AND SHEARBURN E W Jejunal intussusception Surgery 26 833 1949
- HOY C DA COSTA Ileus Internat Clin 4 263 1937
- HUBLIN H Ileus after stomach operations Acta chir scandinav 101 228 1951
- HUGHES M A case of *Bilharzia* of the appendix with strangulation of the ileum Brit J Surg 36 428 1949
- HUGHSON W AND SCARFF J E Influence of intra venous sodium chloride on intestinal absorption and peristalsis Bull Johns Hopkins Hosp 35 197 1924
- HLGO F Roentgenological signs of abdominal effusion Acta radiol 5 63 1926
- HULTEN O Beitrag zur Rontgendiagnose der akuten Pankreasaffektionen Acta radiol 9 222 1928
- HUNT C J Correlation of the X ray diagnosis with the operative findings in small intestinal obstruction Radiology 43 107 1944
- HUNT C J The early clinical and radiologic manifestations of small bowel obstruction South Surgeon 12 62 1946
- HUNT C J Early diagnosis and management of small intestinal obstruction Surgery 19 237 1946
- HUNT C J Early indications of adhesive small bowel obstruction Am J Surg 72 865 1946
- HUNT C J Early diagnosis and roentgen manifestations of obstruction of small bowel Arch Surg 57 460 1948
- HUNT C J Surgical treatment of malignant lesions of the colon complicated by inflammatory reaction fixation or obstruction. South Surgeon 15 248 1949
- HUNT C J Acute bowel obstruction Indust Med 19 471 1950
- HUNT C J Early manifestations and radiologic indications of small bowel obstruction South M J 43 469 1950
- HUNT C J Surgical decompression of the colon for malignant obstruction Arch Surg 61 131 1950
- HUNT V C Intestinal obstruction Southwestern Med 20 167 1936
- HUNTER J A case of paralysis of the muscles of deglutition cured by an artificial mode of conveying food and medicines into the stomach *The Complete Works of John Hunter* edited by J F Palmer vol 3 p 580 Barrington and Haswell Philadelphia 1841
- HUNTER J Proposals for the recovery of persons apparently drowned *The Complete Works of John Hunter* edited by J F Palmer vol 4 p 185 Haswell Barrington and Haswell Philadelphia 1841
- HUNTER J *The Works of John Hunter* ed by James Palmer Longman Rees Orme Brown Green and Longman London 1737
- HURST A F Treatment of achalasia of the cardia Lancet 1 667 1927
- HUSCHKE E *Encyclop die Anatomique* vol 5 p 191 Bailliere Paris 1845
- HUTCHINSON J A successful case of abdominal section for intussusception with remarks on this and other modes of treatment Med Chir Tr London 57 3 1874
- IACNOI Z AND TIMUS C Transmesenteric hernias pathogenesis of mesenteric holes J de chir 50 203 1937
- ICHLER S AND MOLT W F Severe injury to larynx resulting from indwelling duodenal tube cases Ann Otol Rhin & Laryng 48 886 1939
- IMPINK R R AND CLAMMER G R Atresia of the duodenum Ann Surg 116 334 1942
- INGLEFINGER F J Intermittent volvulus of the mobile cecum Arch Surg 45 156 1942
- INGLEFINGER F J Medical progress modification of intestinal motility by drugs New England J Med 229 114 1943
- IVA A C *Digestive System* 2nd ed chap 10 Problems of Ageing pp 254-301 1942
- JABOULAY M La gastro enterostomie La jejunostomie La resection du pylore Arch prov de chir 1 1 1892
- JACKSON C AND JACKSON C L *Diseases of the Anus and Food Passages of Foreign Body Origin* W B Saunders Philadelphia 1936
- JACKSON E W Adynamic ileus in abdominal surgery Kentucky M J 32 284 1934
- JACKSON J A AND EWELL G H Acute intestinal obstruction due to a large gallstone report of a case Wisconsin M J 29 637 1930
- JACKSON J N Membranous pericolicitis and allied conditions of the ileocecal region Ann Surg 57 374 1913
- JACKSON W P U, AND FINDER C C Small gut intussusception following intestinal surgery I A clinical and metabolic study of a man surviving with seven inches of small intestine South African J Clin Sc 2 70 1951
- JACOBS W F Intussusception Illinois M J 103 250 1953
- JACQUES L, DROEGEMUELLER W A AND BUCHHEIMER J R The viability of strangulated intestinal loops Surg Gynec & Obst 55 559 1932
- JAMES T J Retroperitoneal hemorrhage Lancet 2 1123 1930
- JAMES L Intestinal obstruction due to impacted gall stones Australian & New Zealand J Surg 23 77 1953
- JARRF H A Personal communication
- JAWORSKI W Vergleichend experimentelle Untersuchungen ueber das Verhalten Des kessinger an l Karl bader Wassers Arch klin Med 35 38 1884

- JEFFERY L. The ancient Romans through medical eyes *M J Au tralia* 2 419 1934
- JEFFERS H. Pigmentation of the skin *New England J Med* 231 88 1944
- JEFFERS H, MCHUSICK V A AND KATZ K H. Generalized intestinal polyposis and melanin spots of the oral mucosa lips and digits *New England J Med* 241 993 1949
- JENSON J. Atresia of the ascending colon *Lancet* 2 61 1951
- JENKINS C B. Medicine the science and the art *Diplomate* March 1937
- JENKINSON D L AND BATE I C. Volvulus of the stomach *Am J Roentgenol* 69 24 1933
- JENSON H. Decompression treatment with Miller Abbott tube in paralytic ileus *Nord med.* 37 189 1948
- JENSON H J AND FIROR W M. Toxins of high intestinal obstruction. *Am J Surg* 13 281 1931
- JESSETT F B. A suggestion for the treatment of irreducible intussusception of the bowels *Lancet* 1 223 1897
- JOHNER T. Duodenalstenose durch Pancreas anulare *Schweiz. med. Wchschr.* 82 1060 1952
- JOHNSON H T, CONN J W, FIDY A AND COLLIER F A. Postoperative salt retention and its relation to increased adrenal cortical function *Ann Surg* 132 374 1950
- JOHN ON J A. Acute mechanical obstruction of the lower bowel *Minnesota Med* 14 635 1931
- JOHNSON W A AND STRAUSS F H. Intussusception of the small intestine produced by metastatic renal carcinoma *Surgery* 32 991 1952
- JOHNSTON C G. Decompression in the treatment of intestinal obstruction *Surg Gynec. & Obst* 70 365 1940
- JOHNSTON C G. Technique of intestinal intubation vol 5 chap 14 page 651 *Nelson's Loose Leaf Surgery*. Thomas Nelson and Sons New York 1941
- JOHNSTON C G AND CROWLEY R T. Physiological principles in intestinal obstruction *S Clin North America* 26 1427 1946
- JOHNSTON C G, PENBERTH G C AND KENNING J C. Decompression of small intestine in treatment of intestinal obstruction *JAMA* 111 1365 1938
- JOHNSTONE P N, CLASEN A C AND ORR T G. The role of the external secretion of the pancreas in experimental high intestinal obstruction *Surg Gynec. & Obst* 57 483 1933
- JOLLEY A. The diagnosis of intestinal obstruction in the newborn *Brit J Surg* 40 201 1952
- JONES C M AND EATON F B. Postoperative nutritional edema *Arch Surg* 27 159 1933
- JONES C M AND PIERCE F D. Mechanism and reference of pain from the lower intestinal tract *Tr A Am Physicians* 46 311 1931
- JONES G F AND SETTLE J W JR. Obstructing lesion of colon due to non penetrating trauma of the abdomen. *Northwest Med* 51 317 1952
- JONES T L. Surgical treatment of acute and chronic intestinal obstruction *S Clin North America* 17 1409 1937
- JORDAN G L JR AND BEAVERS O H. Volvulus of the cecum as a postoperative complication. *Ann Surg* 137 235 1953
- JORDAN S M. An interesting case of intestinal obstruction *S Clin North America* 11 337 1931
- JORGENSEN M B. Recurrent appendicitis in each trimester of pregnancy *Am J Surg.* 84 331 1952
- JORGENSEN M W, DIETZ N AND HILL F C. Potassium as a toxic factor in intestinal obstruction *Proc Soc Exp Biol & Med.* 43 282 1940
- JOSEPH F G. A new treatment for acute dilatation of the stomach *Am J Surg.* 60 381 1943
- JORDAN F AND HUTER G. Pyloric functioning *Arch. mal. app digest.* 42 265 1953
- JUDD E S AND PUESTOW C B. Chronic duodenal obstruction. *S Clin North America* 13 807 1933
- JUDD J R. Mesenteric defects with special reference to their etiology and report of a rare case of colonic obstruction *Surg Gynec. & Obst.* 48 264 1929
- JUNGMAN H. Volvulus of the caecum *Brit J Radiol* 21 346 1948
- JULIAN H G. Gallstone intestinal obstruction. *Am J Surg* 19 456 1933
- KAHLE H R. Analysis of 151 cases of intussusception from Charity Hospital of Louisiana at New Orleans *Am J Surg* 52 215 1941
- KAHLE H R AND MAES L. Intestinal obstruction—a consideration of the clinical problem. *Mississippi Doctor* 19 114 1941
- KARIN S G, BRIELE H A AND DOUGLAS L H. Volvulus complicating pregnancy *Am J Obst & Gynec.* 43 398 1944
- KALFLEISCH W K. The diagnosis of intestinal obstruction by roentgen ray *Am J M Sc* 174 500 1927
- KALIMA T. Nach Gastroenterostomen und Magenresektionen auftretende Komplikationen. *Duodecim* 42 459 1926
- KALLIO K B. Über Volvulus Coli transversi *Acta chir scandinav* 70 39 1932
- KAMPMIERS R H. Aneurysm of the abdominal aorta—a study of 73 cases *Am J M Sc.* 192 97 1936
- KANTOR J L. A study of atmospheric air in the upper digestive tract *Am J M Sc.* 155 829 1918
- KANTOR J L. Common anomalies of duodenum and colon their practical significance result of 8 years combined clinical and roentgen study *JAMA.* 97 1485 1931
- KAPLE O. Operative treatment of gallstone ileus without enterotomy *Acta chir scandinav* 95 54 1947
- KAPLAN I AND FELCHTWANGER M. Recurrent intussusception due to polyposis of the gastro-intestinal tract associated with pigmentation *Brit M J* 1 659 1953
- KAPLAN I W AND MICHEL M L. Treatment of postoperative adynamic ileus and obstruction of the small

- bowel with the Miller Abbott tube New Orleans M & S J 93 558 1941
- KAPLAN L Congenital duodenal obstruction due to atresia of the duodenum Am J Surg 79 452 1950
- KAPFIS M Erfahrungen mit Lokalanästhesie bei Bauchoperationen Verhandl deutsch & Gesellsch Chir 43 87 1914
- KARABIN J E Retroperitoneal hemorrhage Am J Surg 56 471 1942
- KARN H M Mesenteric vascular occlusion New Zealand M J 52 289 1953
- KAUFFMAN R R AND GERBODE F Arteriomesenteric duodenal ileus Stanford M Bull 9 262 1951
- KAUFMAN L R SERPICO S AND MERSHEIMER W Rare complication of Miller Abbott tube Am J Surg 57 173 1942
- KAUFMAN R L AND VON SAAI F Leukocyte changes in acute peritoneal irritation J Lab & Clin Med 26 468 1940
- KEEFE J W Volvulus of the large and small intestine Am J Surg 17 345 1932
- KEELEY J L Intussusception associated with aberrant pancreatic tissue Arch Surg 60 691 1950
- KEELS L B Gangrenous ileocolic intussusception a case report Mil Surgeon 96 520 1945
- KEENE R Case of acute obstruction at site of gastrojejunostomy Brit J Surg 12 791 1925
- KEHR cited by HELLENS A V AND NUMMI P Spontaneous obstruction of gastroenterostomy and complete detachment of intestinal loop Ann chir et gynaecc Fenniae 40 121 1951
- KELLEY J F Partial intestinal obstruction Am J Surg 22 299 1933
- KELLOGG E L Diagnosis and treatment of chronic duodenal obstruction Surg Gynec & Obst 28 174 1919
- KELLOGG E L AND KELLOGG W A Chronic duodenal obstruction with duodenojejunostomy as a method of treatment Ann Surg 73 578 1921
- KENDALL F Rare case of intestinal obstruction Brit J Surg 18 168 1930
- KENNEDY C S REYNOLDS R P AND CANTOR M O A study of the gastric stoma after partial gastrectomy Surgery 22 41 1947
- KENNEDY C S REYNOLDS R P AND CANTOR M O Paralysis of distal jejunal loop after partial gastrectomy J Michigan M Soc 49 797 1950
- KERR W G AND KIRKALDY WILLIS W H Volvulus of the small intestine Brit M J 1 799 1946
- KETTFVANG E L AND PALUMBO L Arteriomesenteric duodenal compression Arch Surg 62 145 1951
- KEYES E I AND MIDDLEMAN I C The treatment of fistula and obstruction of the small intestine by complete exclusion Surg Gynec & Obst 72 237 1941
- KIERMAN P C AND CLACFETT O T Volvulus of the ileum report of case Mayo Clin Proc Staff Meet 17 446 1942
- KILFOY F J An unusual case of paralytic ileus following removal of cataract Am J Surg 45 137 1939
- KIM S Gastric secretion during high intestinal obstruction Korean M J 2 86 1932
- KINDER C H A case of spontaneous rupture of the inferior epigastric artery simulating acute intestinal obstruction Brit J Surg 40 88 1952
- KINI M G AND VENKOB RAO D Volvulus as a cause of intestinal obstruction Indian M Gaz 73 471 1938
- KIRBY F J Volvulus of cecum Ann Surg 89 475 1929
- KIRKLIN B R A roentgenologic consideration of duodenitis Radiology 12 377 1929
- KIRKLIN B R AND HARRIS M T Hypertrophy of the pyloric muscle of adults a distinctive roentgenologic sign Am J Roentgenol 29 437 1933
- KIRKLIN B R AND WEBER H M Roentgenologic diagnosis of neoplastic diseases of stomach Am J Cancer 16 1134 1932
- KIRKMAN N F BARLOW T AND MOTTERTHEAD S A case of persistent intussusception in an adult Brit J Surg 28 426 1941
- KIRSCHBAUM J D Submucous lipomas of the intestinal tract as a cause of intestinal obstruction Ann Surg 101 734 1933
- KITTLE C F JENKINS H P AND DRASTEDT L R Patent omphalo mesenteric duct and its relation to the diverticulum of Meckel Arch Surg 54 10 1947
- KLECKNER H S JR Chronic ulcerative colitis and pregnancy Am J Obst & Gynec 62 1234 1951
- KLEIN B Duodenal tube applied in treatment for expulsion of tapeworm Arch Schiffs u Tropen Hyg 30 250 1926 abstr JAMA 87 286 1976
- KLEIN P Fatal ileus from contrast suspensions Med Klin 19 829 1923
- KLEIN S H Peroral intubation and drainage of small intestine Technique and indications Surgery 4 827 1934
- KLEINERMAN J YARDUMIAN H T PITTSBURG K AND TAKEWA H T Primary carcinoma of duodenum Ann Int Med 32 451 1950
- KLFINSASSER I J The complications of gastric and intestinal intubation Amer Irac & Digest Treat 2 221 1951
- KLFITSCH W P AND ISNER H G Complete non-rotation of the gut and torsion obstruction of the colon in an adult Am J Digest Dis 21 123 1944
- KLOIBER H Die Roentgendignose des Ileus ohne Kontrastmittel Arch klin Chir 112 513 1919
- KNIGHT G S AND SLOWIE D Strangulation Brit J Surg 23 209 1936
- KOHN S G BRIELE H A AND DOUGLAS L H Volvulus complicating pregnancy Am J Obst & Gynec 48 398 1944
- KOLIMA T Nach Gastroenterotomie und Magenresektionen auftretende Komplikationen Zentralorgan Gesamte Chir., 40 800 1974
- KOLOW A I A new material and tube design in gastrointestinal intubation Surgery 23 293 1948
- KREMAN A J Surgical physiology of intestinal obstruction S Clin North America 29 177 1947

- KRUE, I. G. Duodenal dysplasia. *Ann Surg* 106 33 1937
- KRUEGER, H., ARBUTT, W. I., LEVY, S., HARR, I. I. AND HODGES, W. D. Metabolic alterations in surgical patients: the influence of peritumors on nitrogen, carbohydrate, electrolyte and water balance. *Surgery* 36 580 1954
- KUEHNER, H. G. The management of acute intestinal obstruction. *Lancet* M. J. 54 641 1951
- KUHN, I. Sondierungen am magen Iylorus und Duundarm des men elien. *Arch f Verdauungshw* 3 19 1897
- KUZZ, H. Aufsteigen le Duundarmmagen nach to taler Magenresektion. *Zentralbl Chir* 10 802 1943
- KUNTZ, A. *The Autonomic Nervous Syst m Lea and Feibiger*. Philadelphia 1945
- LACE, T. Gall tone ileus. *Am J Surg* 74 86 1947
- LADD, W. E. Progress in the diagnosis and treatment of intussusception. *Boston M S J* 168 542, 1913
- LADD, W. E. Congenital obstruction of the duodenum in children. *New England J Med* 206 277 1932
- LADD, W. E. Congenital obstruction of the small intestine. *JAMA* 101 1453 1933
- LADD, W. E. AND GRO, R. I. *Ibd minal Surgery of Infancy and Childho t W. I. Saunders Co*. Philadelphia, 1948
- LAKE, M. Influence of weight of duodenal tube tip on its entrance time. *Am J Digest Dis* 7 136 1940
- LAN, H. C. Volvulus of the sigmoid. *Chinese M J* 60 294 1941
- LANDSBERG, D. Ascariasis causing acute intestinal obstruction. *Brit M J* 2 461 1946
- LANGE, K., AND BOYD, L. J. The ue of fluoroscein to determine the adequacy of the circulation. *M Clin North America* 26 943 1942
- LANCE, M. J. An unusual case of intestinal obstruction. *Brit M J* 1 344 1948
- LANGE, cited by TREVES, F. *Intestinal Obstruction Varieties and Their Pathology, Diagnosis and Treatment*. Wm Wood and Co. New York 1899
- LANS, H. S., STEIN, I. F. JR. AND MEYER, K. A. Flec trolyte abnormalities in pyloric obstruction resulting from peptic ulcer or gastric carcinoma. *Ann Surg* 135 441 1952
- LARREY, D. J. *Memoires de chirurgie militaire et cam pagnes* 1812 vol 2 p 146. J. Smith Paris 1812-17
- LASHMET, F. H. AND NEWBERG, L. H. A comparative study of the excretion of water and solid by normal and abnormal kidneys. *J Clin Invest* 11 1003 1932
- LATIMER, H. B. A towel found in the jejunum. *J Kansas M Soc* 53 516 1952
- LATOUR, A. Considerations pratiques sur les cau es la nature le diagnostic et le traitement des gas developpes dans les voies digestives. *Bull gen de therap* Paris 30 338 1846
- LAUFMAN, H. Experimental evidence of factors concerned in the eventual recovery of strangulated intestine: effects of massive penicillin therapy. *Surgery* 28 509 1950
- LAUFMAN, H. AND FREY, S. C. Observations on the firm white int intinal strangulation. The effect of adrenal cortical extract on it tivity. *Surg Gyneec & Ol* 1 77 605 1947
- LAUFMAN, H., MARTIN, W. B., METHOD, H., TUELL, S. W. AND HARDING, H. Observations in strangulation obstruction: The fate of sterile devascularized intestine in the peritoneal cavity. *Arch Surg* 59 550 1949
- LAUFMAN, H. AND METHOD, H. The role of vascular spasm in recovery of strangulated intestine. *Surg Gyneec & Obst* 85 675 1947
- LAUFMAN, H., TANTURI, C. A. AND FLEUR, W. F. JR. Attempts at detection of lecithinase in the blood and lymph following intestinal strangulation obstruction. *Surg Gyneec & Obst* 93 292 1951
- LAUBALL, H. F. Acute abdominal cases chiefly from the roentgenological point of view. *Acta chir scandinav.*, 66 105 1930
- LAWEN, A. Zur Operation des ileus. *Zentralbl Chir* 54 1037 1927
- LAWSO, E. H. AND WHITENER, D. L. Retrograde jejuno gastric intussusception. *Arch Surg* 60 242 1950
- LAYLOCK, H. T. Irreducible intussusception in a baby treated by resection. *recovery*. *Brit M J* 1 120 1941
- LAZARUS, J. A. AND ROSENTHAL, A. A. Intestinal obstruction. *Am J Surg* 13 490 1931
- LEAF, A. AND MAMBY, A. Antidiuresis mechanism not regulated by extracellular fluid tonicity. *J Clin Invest* 31 60 1952
- LEE, C. M. JR. Intestinal obstruction in the newborn infant. *J M A Georgia* 41 233 1952
- LEFF, C. M. JR. High intestinal obstruction in newborn and very young infants. *J Kentucky M A* 51 277 1953
- LEE, M. Intestinal obstruction by gallstones. *Brit M J* 1 555 1945
- LEICHTENSTERN, O. *Cyclo dia of Practice of Medicine* vol 7 p 569. Ziemssen New York Wm Wood and Co. New York 1876
- LEICHTENSTERN, O. *Handbuch der Specellen Pathologie und Therapie*. F. C. W. Vogel Leipzig 1876
- LEIGH, O. C. JR. Ileus associated with edema of the bowel. *Surg Gyneec & Obst* 75 279 1942
- LEIGH, O. C. JR., NELSON, J. A. AND SWENSON, P. C. The Miller Abbott tube as an adjunct to surgery of small intestinal obstructions. *Ann Surg* 111 186 1940
- LEITCH, P. Duodenal atresia in a newborn infant. *Brit M J* 2 885 1945
- LEITHAUSER, D. J. Simplified suction unit for intestinal decompression. *JAMA* 127 157 1945
- LEITHAUSER, D. J. Atypical adynamic ileus apparently caused by nutritional (thiamine chloride) deficiency. *Surg Gyneec & Obst* 86 543 1948
- LEITHAUSER, D. J. AND CANTOR, M. O. Intestinal obstruction caused by towel. Unpublished data
- LENNANDER, K. G. Ein Fall von Duundarmvolvulus mit einen Meckelschen Divertikel. nebst einigen Worten uer

- sub iléon Ileus und über Gastrotomie bei Dünndarm
paralyse Deut che Zt chr Chir 86 1 1907
- JENNON C The rare causes of abdominal pain in preg-
nancy Proc Pos Soc Med 43 105 1950
- LENORMANT C AND CORBIER G Du ballonnement ab-
dominal sans les hémorrhages sous-péritoneales Presse
med 42 1257 1934
- LEVYARD T B Intestinal obstruction caused by preg-
nancy Virginia M Monthly 22 30 1917
- LEVIN A L Transduodenal lavage and usefulness of
the Jutte tube South M J 13 490 1920
- LEVIN A L New gastroduodenal catheter JAMA
76 1007 1921
- LEVITIN J Ileocolic intussusception diagnosis by x ray
without contrast media Am J Surg 54 494 1941
- LEWIS E E A case of retrograde intussusception occur-
ring during life Brit J Surg 23 683 1936
- LEWIS F J Intestinal atresia in identical twins Sur-
gery 33 121 1953
- LEWIS K M Acute mechanical intestinal obstruction
caused by acute appendicitis Ann Surg 105 291 1937
- LICHTENSTEIN M F The basis for therapy in intestinal
obstruction Illinois M J 81 309 1942
- LICAT D AND OVEREND T D Recurrent volvulus of
the pelvic colon Brit M J 2 7 1933
- LICHT R A The nature of the toxic material in closed
loop intestinal obstruction identification of lysozyme
Surgery 30 195 1951
- LINDENMUTH W W Fecal fistula due to metallic mer-
cury from a Miller Abbott tube JAMA 141 986
1949
- LINDQUIST J L Physiological problems in suction drain-
age of the gastrointestinal tract Illinois M J 81 49
1942
- LIPPERANCE cited by TREVVS F Intestinal Obstruc-
tion Varieties and Their Pathology, Diagnosis and
Treatment Wm Wood and Co New York 1899
- LIPPINCOTT S W A case of massive volvulus of the
sigmoid with unusual gut distention Canad MAJ 37
489 1937
- LIPPMAN C W Simplification of the duodenal tube ex-
amination JAMA 62 911 1914
- LITTLÉ A cited by BROWNLOW C V Gould's Medical
Dictionary The Blakiston Co Philadelphia 1945
- LOBINGER A S Acute obstruction of the jejunum
caused by a fibrous bolus S Clin North America 10
1099 1930
- LOCKHART MUMFERY J P Acute obstruction of the
colon Brit M J 1 405 1933
- LOCKWOOD A L Acute intestinal obstruction involving
the appendix Canad MAJ 23 400 1930
- LOCKWOOD C D Major surgery in people of advanced
age S Clin North America 13 105 1933
- LOCKWOOD J S AND RANDALL H T The place of
electrolyte studies in surgical patients Bull New York
Acad Med 25 228 1949
- LOEB M J Mesenteric cysts a review of literature
genesis and classification Report of a case New York
J Med 41 1564 1941
- LOFFELER L Ein Beitrag zur Morphologie der Gallen-
steine mit Hilfe der Roentgenstrahlen Beitr path
Anat 78 44 1927
- LOFSTROM J E AND NOER R J The role of inte-
stinal intubation in the localization of lesions of the gastro-
intestinal tract Am J Roentgenol 42 321 1939
- LOFSTROM J S AND NOER R J The role of intestinal
intubation in the diagnosis and localization of intestinal
obstruction Radiology 35 546 1940
- LONG F R Acute intestinal obstruction in a newborn
infant from hernia of the lower ileum through a con-
genital mesenteric opening Tr Chicago Path Soc 12
335 1927
- LONG J W The value of enterostomy in intestinal ob-
struction Texas J Med 18 606 1973
- LONG L D The prevention and treatment of post op-
erative intestinal incompetence (paralytic ileus) South
M J 26 350 1933
- LOPES PONTES J P Simple recourse for accelerating
intubation Rev brasil med 1 941 1944
- LORD J W JR HOWES E L AND JOLLIFF N The
surgical management of chronic recurrent intestinal
obstruction due to adhesions Ann Surg 129 315 1949
- LORETZ W Ein Fall von gangliosem Neurom Arch
path Anat 49 435 1870
- LOWDON A G P Deflation of distended bowel at opera-
tion Lancet 1 1103 1951
- LOWMAN R M AND WISSING F C Preoperative
roentgen diagnosis of gallstone ileus JAMA 112
2247 1939
- LUDWIG I E AND LUDWIG C A Intra uterine vol-
vulus of the ileum J Michigan M Soc 48 858 1949
- LUNDY J S Anesthesia and supportive therapy for
operations on elderly patients with special reference to
reduction of operative risk Geriatrics 3 361 1948
- LURTON C H Intestinal obstruction Virginia M
Monthly 66 610 1939
- LURTON C H Intestinal obstruction Am J Surg 83
794 1952
- LYALL A Prevention of peritoneal adhesions by corti-
sone Glasgow M J 34 208 1933
- LYDAY R O Congenital duodenal obstruction Am J
Surg 54 678 1941
- MA A C AND SLY AND STEIN C C Intussusception
an analysis of twenty eight cases Chinese M J 69
239 1951
- MACDONALD K C Congenital volvulus of the small
intestine West Virginia M J 48 285 1952
- MACFARLANE M B AND KNIGHT B C Biochemistry
of bacterial toxins lecithinase activity of Clostridium
tetani toxin Biochem J 35 884 1941
- MACFARLANE J A AND KAY S K Organisms synthe-
sizing false colonic obstruction Is it a new entity? Brit M
J 4639 1967 1949

- MAILEY W I Mechanical obstruction of the small intestine New York J Med 40: 424 1940
- MACKENZIE K C I An unusual case of ileocolic obstruction Brit M J 1 931 1948
- MACKENZIE J J, SCHWARTZ I I AND ROBERTAZZI R W Intestinal hernia causing acute intestinal obstruction Am J Surg 88 354 1944
- MACKEY T T Vitamin deficiencies and small intestine JAMA, 117 910 1941
- MACLENNAN D Hemorrhage from deep epigastric artery into rectus abdominis Brit M J, 1 895 1928
- MACNAB D S Intussusception Canad MAJ 42 447 1940
- MACPHAIL A The source of modern medicine an address to the American College of Physicians February 8 1933
- MADDERN An account of what was observed upon opening a person who had taken several ounces of crude mercury internally and a plumb-stone lodged in the coats of the rectum Phil Tr Roy Soc London 44 152 1732
- MADDING C F, IVERIART M W AND HEATH H J Congenital duodenal atresia with malrotation of the intestine Ann Surg, 131 433 1950
- MADDOCK W C, BELL J I AND TREMAINE M J Gastrointestinal gas observations on belching during anesthesia, operations and pyelography and rapid passage of gas Ann Surg 130 512 1949
- MAFS U A surgical consideration of appendicitis in pregnancy Am J Obst & Gynec 27 214 1934
- MAGNEY F H Acute intussusception in infancy and childhood. Minnesota Med 30 257 1947
- MACNEILSON W Meteorism in pyelography and on the passage of gas through the small intestine Acta radiol 12 552 1931
- MACLEAF C H Obstruction of the gastrointestinal tract in infants South Surgeon 16 144 1950
- MAION G D Use and abuse of the indwelling tube in abdominal surgery Dallas M J 30 96 1944
- MAHONEY F B, AND SHERMAN C D JR. The management of acute small bowel obstruction New York J Med 51 370 1951
- MAHONEY L F AND BLUM J W Multiple intestinal obstruction Am J Surg, 22 312 1933
- MAHORNOR, H Matting syndrome a type of intestinal obstruction a review of 60 cases of intestinal obstruction. New Orleans M & S J., 104 17 1951
- MAIER R Beitrage zur angeborenen pylorusstenose Arch Path Anat 102 413 1885
- MAINGOT R Abdominal Operations 2nd ed D Appleton Century Co New York 1948
- MAINZER F S Acute obstruction of the large intestine Penn M J 34 251 1931
- MAJOR R D The papyrus ebers Ann M Hist 2 547 1930
- MALL, G P A study of the causes underlying the origin of human monsters J Morphol 19 1 1908
- MALT I I On the frequency of localized anomalies in human embryos and infants at birth Am J Anat 22 41 1917
- MALLET C L AND BELLIERE Radiologic exploration of anastomotic openings of cholecystogastric stomy Arch mal app digest 16 686 1926
- MALLOREY T B Case Report # 33702 New England M J 236 763 1947
- MALLOY S R Factors inducing renal shut-down from lysed erythrocytes an experimental study Ann Surg 130 39 1949
- MANSLEY J F Water and electrolyte balance Physiol Rev 34 334 1954
- MANTREDI D H Rational therapeutics of intestinal obstruction in infancy New York J Med 49 1691 1949
- MANTREDI D H Cholelithic intestinal obstruction U S Armed Forces M J 2 243 1951
- MANON L Volvulus of the cecum Am J Surg 87 264 1954
- MARBURY W B Adynamic ileus Internat J Med & Surg 48 250 1955
- MARBURY W B Discussion of paper by DAVID A C, AND GILCHRIST R K Extension of the border line of operability in cancer of the rectum Tr South S A 54 92 1941
- MARONI A Varieta rarissima di occlusione acuta del tenue da diverticolo strozzamento rotatorio coesistente nello stesso intestino Morgagni 29 273 1887
- MARSHAK R H, LESTER L J AND FRIEDMAN A I Megacolon—a complication of ulcerative colitis Gas troenterology 16 768 1950
- MARTIN C H Congenital anomalies of the gastrointestinal tract in infants and children South Surgeon 15 163 1949
- MARTIN I AND WRIGHT H E Low grade partial obstruction of the small intestine Bull Johns Hopkins Hosp 62 477 1938
- MARTINOTTO G L'intubazione intestinale quale trattamento pre e postoperatoria nello chirurgia del retta Arch ed Atti della Soc eta Italiana di Chirurgia 2 1 1950
- MARTINOTTO G Splanchnic nerve block as an adjuvant in intestinal intubation J Internat Coll Surgeons 18 881 1952
- MARTZLOFF K H Prolapse of the intestine through a preformed opening in the great omentum Surg Gynec & Obst 50 899 1930
- MASON A R Intestinal obstruction some medical aspects Calif & West Med 32 1 1930
- Massachusetts General Hospital Case # 33202—Callstone Ileus New England J Med 236 763 1947
- MASSFENCILL S C A Sketch of Medicine and Pharmacy The S F Massengill Co Bristol Tenn 1940
- MAST W H Recurrent intestinal obstruction due to gallstones Am J Surg 32 516 1936
- MASTIN E V Multiple attacks of acute intussusception Am J Surg 58 300 1947

- MAYAS R Continued intravenous drip with remarks on value of contained gastric drainage and irrigation by nasal intubation with gastroduodenal tube (Jutte) in surgical practice *Ann Surg* 79 643 1924
- MATEER J G Intestinal obstruction *Am J Surg* 12 89 1931
- MATTHEWS F S John of Arderne *Bull New York Acad Med* 6 461 1930
- MATIGNON A Du traitement de l'occlusion intestinale par le mercure metallique a haut dose These de Paris 340 1 1870
- MATTISON J A Acute intestinal obstruction *Am J Surg* 7 644 1929
- MAY A M AND TORRE E Fecal impaction report of unusual case *Calif & West Med* 59 224 1943
- MAYER H JR Passage of Miller Abbott tube through the pylorus with aid of electromagnet *US Naval M Bull* 43 463 1944
- MAYO C H AND MAGOUN J A N JR Postoperative intra abdominal hernia *Arch Surg* 4 324 1922
- MAYO C W Acute intussusception *Collect Papers Mayo Clin* 24 121 1932
- MAYO C W AND BROWN P W Intestinal obstruction caused by a gallstone *Surgery* 25 924 1949
- MAYO C W MILLER J M AND STALKER L H Acute intestinal obstruction *Surg Gynec & Obst* 71 589 1940
- MAYO C W AND NETTROUR W S Carcinoma of the jejunum *Surg, Gynec & Obst* 65 303 1937
- MAYO C W AND STEINBERG A G Familial incidence of primary carcinoma of jejunum *JAMA* 146 797 1951
- MAYO C W AND WAKEFIELD E G Intestinal obstruction associated with anomalies of rotation and fixation of the intestines report of a case *Am J Surg* 46 373 1939
- MAYO C W AND WOODRUFF R Acute intussusception *Arch Surg* 43 583 1941
- MAYO H W JR Dissecting aneurysm simulating large bowel obstruction *Am J Surg* 83 522 1951
- MCDAMIS A J An analysis of causes of morbidity and mortality in colonic surgery *Am J Surg* 86 530 1953
- MCCALL M L Surgical complications of pregnancy *S Clin North America* 28 1507 1948
- MCCAGHAN J M AND COUGHLIN W T Posterior gastrojejunostomy *Surg Gynec & Obst* 65 824 1937
- MCCLURE R D An experimental study of intestinal obstruction *JAMA* 49 1003 1907
- MCCOLLUM E B Melanotic metastatic sarcoma of the small bowel *Grace Hosp Bull* 32 41 1954
- MCCORMICK E J Incidence and mortality of intestinal obstruction *Ohio M J* 26 755 1930
- MCCREADY F J AND SEYBOLD W D Intestinal obstruction caused by gallstone *Mayo Clin Proc Staff Meet* 23 579 1948
- MCCUNE W S AND KESHISHIAN J M Postoperative intestinal obstruction *Surg Gynec & Obst* 96 567 1953
- MC EVERS A E Acute intestinal obstruction complicating thyrotoxicosis *Am J Surg* 40 662 1938
- MCGLENNAN A An address on acute intestinal obstruction *South Med & Surg* 92 221 1930
- MCGRAW J P KREWEY A J AND RIGLER L G The roentgen diagnosis of volvulus of the cecum *Surgery* 24 793 1948
- MCGUFF P WAUGH J M DOCKERTY M B AND RANDALL L M Endometriosis as a cause of ileal obstruction *Am J Obst & Gynec* 56 1059 1948
- MCGUILL P DOCKERTY M B WAUGH J M AND RANDALL L M Endometriosis as a cause of intestinal obstruction *Surg Gynec & Obst* 85 273 1948
- MCIVER M A Acute intestinal obstruction I General considerations *Arch Surg* 25 1098 1932
- MCIVER M A Acute mechanical obstructions exclusive of those due to neoplasms and strangulated external hernias *Arch Surg* 25 1106 1932
- MCIVER M A Obstruction due to neoplasms and strangulated external hernia *Arch Surg* 25 1125 1932
- MCIVER M A Acute mechanical obstruction *Am J Surg* 19 365 1933
- MCIVER M A Acute intestinal obstruction neoplasms *Am J Surg* 20 171 1933
- MCIVER M A *Acute Intestinal Obstruction* Paul B Hoeber Inc New York 1934
- MCIVER M A REDFIELD A C AND BENEDICT F B Gaseous exchange between blood and lumen of stomach and intestines *Am J Physiol* 76 92 1926
- MCKECHNIE P E AND PRIESTLEY J T Volvulus of small intestine *Am J Surg* 34 286 1936
- MCKENNA H Intestinal obstruction *S Clin North America* 24 146 1944
- MCKENZIE A D MOOR J R AND MILLER G G Complications of gastrointestinal intubation *Canad M A J* 67 403 1952
- MCKITTRICK L S The diagnosis and management of acute obstruction of the small intestine *New England J Med* 225 647 1941
- MCKITTRICK L S AND SARRIS S P Acute mechanical obstruction of the small bowel Its diagnosis and treatment *New England J Med* 222 611 1940
- MCKITTRICK L S AND WARREN P The use of the Miller Abbott tube in sub-total colectomy and other surgical procedures *JAMA* 117 345 1941
- MCLAUGHLIN C W JR Acute intussusception in infancy and childhood *Am J Surg* 76 306 1943
- MCLAUGHLIN C W JR Surgical management of irreducible intussusception *Arch Surg* 56 48 1948
- MCLAUGHLIN C W AND BRUSH J H Factors responsible for improved results in the management of acute intestinal obstruction *Arch Surg* 61 115 1950
- MCLAUGHLIN C W JR AND BRUSH J H The surgical management of acute intestinal obstruction *Nebraska M J* 33 51 1948

- McLAUGHLIN C W AND RAINES M Obstruction of the alimentary tract from gallstones Am J Surg 81 424 1911
- McFAN C C Acute obstruction small intestine general considerations Kentucky M J 36 226 1938
- McFAN D AND ARMINSKI T Personal communication
- McLEAN E H Intestinal obstruction Northwest Med 31 193 1932
- McMILLAN W M AND MAON R H Pseudocyst in the transverse mesocolon producing obstruction of the duodenum at the ligament of Treitz Am J Surg 84 496 1952
- McNAMARA J P FARBER L A AND NESELER A B Acute intestinal obstruction due to an impacted gall stone J Iowa M Soc 26 45 1936
- McNAUGHT J B Annular pancreas a complication of 40 cases with a report of a new case Am J M Sc 185 249 1933
- McNEALY J W AND LICHTENSTEIN M F Gastrojejunostomy preoperative decompression Surg Gynec & Obst. 63 96 1936
- McNEALY R W AND LICHTENSTEIN M F Acute mechanical ileal obstruction following appendectomy Am J Surg 55 157 1942
- McQUARRIE L AND WHIPPLE G H Penial function influenced by intestinal obstruction J Exper Med 29 397 1919
- McSWAIN B Intussusception of the appendix South M J. 34 263 1941
- McVICAR C S A discussion of the clinical and laboratory findings in certain cases of obstruction in the upper gastrointestinal tract Am J M Sc 169 224 1925
- McVICAR C S AND WEIR J F Toxemia of intestinal obstruction and ileus clinical deductions regarding its nature and treatment Mayo Clin Proc Staff Meet 3 193 1928
- McWATERS R C Volvulus of small intestine Brit M J 2 776 1945
- McWORTER G L Chronic intermittent obstruction of the ascending colon by parietocolic band or membranes S Clin. North America 16 101 1936
- MEAD W R Intestinal obstruction review of pathological processes underlying certain of the symptoms J South Carolina M A 26 127 1930
- MECKEL J F Ueber die Divertikel am Darmkanal Arch Physiol 9 421 1809
- MEFONCA J Diagnosis and treatment of acute intestinal obstruction. Surg Gynec & Obst 52 1115 1931
- MEILING R L Appendicitis complicating pregnancy labor and the puerperium Surg Gynec & Obst 85 512 1947
- MEISELAS D A A case of gallstone ileus Rev Gastroenterology 14 857 1947
- MELAMED A Volvulus of megacolon reduced during barium enema examination Radiology 45 392 1945
- MELAMED A AND MARCA A Esophageal obstruction due to Serutan J A M A 157 318 1953
- MELLINS H Z AND CANTOR M O Intestinal intubation Bull Sinai Hosp Detroit 3 8 1955
- MELLINS H Z AND MILMAN D H Congenital duodenal obstruction Am J Dis Child. 72 81 1946
- MELLINS H Z AND RIGLER L G Roentgen observations in strangulation obstruction Bull Univ Minn Hosp 24 241 1952
- MENAKER J S AND CAUBLE W G Spontaneous rupture of utero-ovarian veins in pregnancy complicated by massive bowel hemorrhage Obst & Gynec. 2 92 1953
- MENSING F H Intestinal obstruction Am J Surg. 17 206 1932
- MENSING F H Treatment of intestinal obstruction Illinois M J 61 511 1932
- MENSING E H Reducing the hazards in the treatment of intestinal obstruction Wisconsin M J 33 807 1934
- MENTER S H Obstructive cholecystitis with particular reference to acute obstructive cholecystitis and its sequelae Surg Gynec & Obst. 62 879 1936
- MERSHEIMER W L WINFIELD J M AND FRANK HAUSER R L Mesenteric vascular occlusion. Arch Surg 66 752 1953
- METHENY D AND HARTZELL H V Diagnosis and treatment of intestinal obstruction Northwest Med. 41 49 1947
- METHENY D AND HUTCHINS L R The use of gravity and Wangenstein tip in Miller Abbott intubation West J Surg 50 618 1942
- METHENY D AND LUNDMARD V O Mechanism of gas patterns in intestinal obstruction West J Surg 61 566 1953
- METHENY D AND NICHOLS H E Volvulus of the sigmoid Surg Gynec & Obst 76 239 1943
- METRAKOS J D Congenital hypertrophic pyloric stenosis in twins Arch Dis Child. 28 351 1953
- MEYER H W Acute superior mesenteric artery thrombosis Arch Surg 53 298 1946
- MEYER K A AND SINGER H A Intermittent gastric ileus due to mechanical causes Surg. Gynec & Obst 53 747 1931
- MEYER R The pathology of some special ovarian tumors and their relation to sex characteristics Am J Obst & Gynec 22 697 1931
- MICHEL M L JR. Acute malignant obstruction of the large bowel South Surgeon 13 299 1947
- MICHEL M L JR KNAPP L AND DAVIDSON A Acute intestinal obstruction Surgery 28 90 1950
- MICHEL M L JR AND McCAFFERTY E L Acute obstructions of the colon Arch Surg 57 774 1948
- MICHEL M L JR AND McCAFFERTY E L Volvulus of the colon South Surgeon 14 525 1948
- MIGLIACIO A V Extraperitoneal cecostomy Rhode Island M J 26 96 1943
- MIKAL S AND BYERS J A Closed loop obstruction of the ileum due to an appendical knot J A M A 160 49 1956

- MILLER A J Cause of death in intestinal obstruction Kentucky M J 29 183 1931
- MILLER, C J A clinical consideration of intestinal obstruction Am J Surg 8 509 1930
- MILLER E M Bowel obstruction in the newborn Ann Surg 110 587 1939
- MILLER H P Acute intestinal obstruction Illinois M J 58 145 1930
- MILLER J M Volvulus of ileocaecal region Mayo Clin Staff Meet 15 424 1940
- MILLER T G Personal communication to authors at A M A Convention at Atlantic City 1949
- MILLER T G AND ABBOTT W O Intestinal intubation practical technique Am J M Sc 187 595 1934
- MILLER T G ABBOTT W O AND KARR W G Intubation studies of human small intestine miscellaneous observations Am J Digest Dis and Nutrition 3 647 1936
- MILLET J B Cecostomy and the Miller Abbott tube Surg Gynec & Obst 84 1083 1947
- MILLIGAN E T C AND SIMON G Routine radiograms for investigation of intestinal obstruction Brit M J 1 1114 1931
- MIMPRISS T W AND BIRT ST J M C Results of partial gastrectomy for peptic ulcer Brit M J 2 1095 1948
- MITCHELL J K On the penetrativeness of fluids J Roy Inst 2 101 1831
- MOLESWORTH H W L Ileocolostomy report of accident from this operation with some remarks upon results of closed ileal loop in man Brit J Surg 17 344 1929
- MOLFINO F Knotting of duodenal tube as rare accident case Gaz d osp 54 1050 1933
- MOLINOFF M W AND SHLIMBAUM S Intestinal obstruction due to gallstones diagnosed pre operatively New York J Med 50 2197 1950
- MONKS G H Intestinal localization Ann Surg 38 574 1903
- MOURA A K The complications of gastric and duodenal ulcer Postgrad M J 24 177 1948
- MONTENIRO M D AND FILHO M C Thiamine hydrochloride and gastro intestinal motility use in post operative period of abdominal surgery to prevent ileus Hospital Rio de Janeiro 24 601 1943
- MONTGOMERY A H The treatment of intestinal obstruction S Clin North America 20 13 1940
- MOORE F D Adaptation of supportive treatment to needs of surgical patient J A M A 141 646 1949
- MOORE F D The low sodium syndromes of surgery J A M A 154 379 1954
- MOORE, G A Acute intestinal obstruction Rhode Island M J 10 173 1927
- MOORE R M Acute intestinal obstruction in infants and children physiological pathological considerations Mississippi Doctor 23 554 1946
- MORGENSTERN M Simplified procedure for introduction of tube into duodenum J A M A, 97 175 1931
- MORIN G AND VIAL J On the extent of the inhibitory reflex produced by distention of the intestine Com rend Soc de biol 115 1540 1934
- MORITZ A R Developmental anomalies causing or predisposing to intestinal obstruction Ohio M J 30 479 1934
- MORLOCK C G AND GRAY H K Congenital duodenal obstruction Ann Surg 118 372 1943
- MORRIS J H Inflammatory tumors of the gastrointestinal tract Ann Surg 97 889 1933
- MORRIS J H AND JOHNSON V S Hernia as an etiologic factor in acute intestinal obstruction Surgery 1 903 1937
- MORRISON W R High intestinal obstruction caused by primary carcinoma of proximal jejunum Am J Surg 2 154 1927
- MORRISON W W Diseases of Nose Throat and Ear p 381 W B Saunders Co Philadelphia 1939
- MORTOLA G A El meteorismo en la enfermedad post operatoria Prensa med argent 31 1516 1944
- MORTON C B II ALDRICH E M AND HILL L D Internal hernia after gastrectomy Ann Surg 141 759 1955
- MORTON H B Improved tip for Miller Abbott tube Ann Surg 117 159 1943
- MORTON J H FURTH F W HINSHAW J R AND SCHILLING S A The use of Aureomycin in experimental intestinal obstruction Ann Surg 134 1007 1951
- MORTON J J Differences between high and low intestinal obstruction in dog anatomic and physiologic explanation Arch Surg 18 1119 1929
- MORTON J J Treatment of ileus as indicated by clinical experience and experimental studies Ann Surg 95 856 1932
- MORTON J J Atresia of the duodenum Am J Dis Child 25 371 1935
- MORTON J J AND JONES T B Obstructions about the mesentery in infants Ann Surg 104 864 1936
- MORTON J J AND PEARSE, H E JR The similarity in effect of experimental high intestinal obstruction and high complete intestinal fistula Ann Surg 94 263 1931
- MORTON J J AND SULLIVAN W C A comparison between simultaneous equal sized closed obstruction of the duodenum and ileum Arch Surg 21 531 1930
- MOSS W AND McFETRIDGE, E M Acute intestinal obstruction Ann Surg 100 158 1934
- MOUAT T B Stricture of bowel by misplaced endometrial tissue Brit J Surg 14 76 1926
- MOYER C A Fluid and electrolyte balance Surg Gynec & Obst 84 586 1947
- MOYER C A Acute temporary changes in renal function associated with major surgical procedures Surgery 27 198 1950
- MOYER C A Non strangulating intestinal obstruction Texas J Med 46 157 1950

- MOYER C A *First Balance 1 Clinical Manual Year* Book Publishers Chicago 1952
- MOYNIHAN B A On retroperitoneal hernia *The Irris and Cal Lectures* Bailliere Tindall and Cox London 1906
- MOYNIHAN B G A *Abdominal Operations* 4th ed W B Saunders Philadelphia 1926
- MULLER B I Chronic duodenal obstruction two case reports *West J Surg* 47 85 1939
- MURCHISON C *Clinical Lectures on Diseases of the Liver Jaundice and Abdominal Dropsy* 3rd ed Wm Wood and Co New York 1885
- MURPHY F T AND VINCENT B An experimental study of the cause of death in acute intestinal obstruction *Boston M C S J* 145 684 1911
- MURPHY H H Indian medicine *Canad M A J* 17 725 1927
- MURPHY J B Gallstone disease and its relation to intestinal obstruction *Illinois M J* 18 277 1910
- MUSSEL J J Intussusception *S Clin North America* 24 703 1944
- NAFF C A Acute intestinal obstruction *J Indiana M A* 27 293 1934
- NAGEL C F Incomplete duodenal diaphragm as a cause of intestinal obstruction *J Internat Coll Surgeons* 2 315 1939
- NARAT J K AND MANELLI I A First gastrectomy structure of the efferent loop and its treatment *Arch Surg* 66 192 1933
- NEER C S Gallstone ileus *J Oklahoma M A* 25 105 1932
- NELSON A *Clinical Lectures on Surgery from Notes Taken by H F Hille MD* J B Lippincott and Co Philadelphia 1825
- NELSON H Obstruction of the sigmoid complicating a fracture of the pelvis *Journal Lancet* 58 301 1938
- NEMIR P JR Gallstone ileus *Surg Gynec & Obst* 94 469 1952
- NEMIR P JR Intestinal obstruction Ten years statistical survey at the hospital of the University of Pennsylvania *Ann Surg* 135 3 1952
- NICHOLS H G Intussusception in adults *Surg Gynec & Obst* 73 832 1941
- NITKIN R L AND LESSER A Intestinal obstruction due to a gallstone *Ann Surg* 118 101 1943
- NOBLE T P JR Plication of small intestine as prophylaxis against adhesions *Am J Surg* 35 41 1937
- NOBLE T B JR Plication of the small intestine *Am J Surg* 45 574 1939
- NOER R J Intestinal obstruction *J Kentucky M A* 51 448 1933
- NOER R J AND DERR J W Effect of distention on intestinal revascularization *Arch Surg* 59 532 1949
- NOER R J AND JOHNSON C G Small intestine obstruction a five year study *Ann Surg* 115 935 1942
- NOER R J POBB H J AND JACOBSON L F Circulatory disturbances produced by acute intestinal distention in living animals *Arch Surg* 63 520 1921
- NOLAN J OI AND FINLEY G C Aseptic intestinal decompression during surgery *New England J Med* 242 54 1950
- NYGAARD I Bloodless reduction under roentgenologic control of acute volvulus of the sigmoid flexure *Acta chir scandinav* 90 17 1944
- NORRIS E AND CAY R Preoperatively diagnosed ileus due to a gallstone *Acta radiol scandinav* 30 4/9 1948
- NORRIS W J Intestinal obstruction in children *West J Surg* 46 249 1938
- NOSSIN L A AND TANNENBAUM W J Recurrent gallstone ileus case report *Surgery* 31 599 1922
- NYBORG S Intussusception in children a study based on one hundred and eight cases *Acta chir scandinav* 80 1 1943
- NYGAARD K K Intracolonic resection of non reducible intussusception *Am J Surg* 87 589 1954
- OBANNON R P Intestinal obstruction in the newborn *Texas J Med* 38 728 1943
- OCHSNER A Acute intestinal obstruction *South M J* 24 93 1931
- OCHSNER A Acute intestinal obstruction *Surg Gynec & Obst* 52 702 1931
- OCHSNER A X ray diagnosis of ileus value of roentgenograms in simple and strangulated obstruction *Surg Gynec & Obst* 56 719 1933
- OCHSNER A Physiologic considerations of ileus *Am J Roentgenol* 37 433 1937
- OCHSNER A J Constriction of the duodenum below the entrance of the common duct and its relation to disease *Ann Surg* 42 80 1906
- OCHSNER A J cited by GAMBLE H A The role of obstructive enterostomy in the treatment of obstruction of small intestine *South M J* 42 480 1949
- OCHSNER A AND GAGE I M Adynamic ileus *Am J Surg* 20 378 1933
- OCHSNER A GAGE I M AND CUTTING R A The influence of hypertonic salt solution on the motility of normal and obstructed intestine *Arch Surg* 27 747 1933
- OCHSNER A AND STORCK A H Mechanical decompression of the intestine in treatment of ileus effect of stripping on blood pressure *Arch Surg* 33 664 1936
- O'CONNOR J M Fibrosarcoma of the small intestine causing intussusception *M J Australia* 2 491 1946
- OLIVIE H Large intestine colic due to sympathetic deprivation new clinical syndrome *Brit M J* 2 671 1948
- OLDHAM J B Small bowel obstruction following antecolic partial gastrectomy *Brit J Surg* 40 625 1953
- OLIVER J MACDOWELL M AND TRACY A The pathogenesis of acute renal failure associated with traumatic and toxic injury Renal ischemia nephrotoxic damage and the ischemic episode *J Clin Invest* 30 1307 1951
- OLSEN A M HOLMAN C B AND ANDERSEN H A The diagnosis of cardiopneumia *Dis Chest* 23 477 1953

- ORGEL, D H Barium meal simulating intestinal obstruction M J & Rec 129 396 1929
- ORR T G Lethal factors in intestinal obstruction Surg Gynec & Obst 55 383 1932
- ORR, T G AND HADEN R L Water and salt imbalance in high intestinal obstruction and its relation to treatment New York J Med 30 1161 1930
- ORR T G A possible danger of enterostomy in the treatment of intestinal obstruction J Kansas M Soc 34 60 1933
- OSGOOD E C The role of the radiologist in the management of patients with intestinal obstruction with special reference to the use of the Miller Abbott tube Radiology 49 529 1947
- OSLER, W Notes on intestinal diverticula Ann Anat & Surg 4 202 1881
- OTERO J P Post gastrectomy occlusion Bol Soc cir Uruguay 21 20 1950
- OVERGARD A W AND JOHNSON J W Mesenteric vascular occlusion Wisconsin M J 52 541 1953
- OWEN J G Volvulus of the transverse colon a case report Bull Mason Clinic 5 127 1951
- OWENS F M JR Gallstone ileus Gastroenterology 1 938 1943
- PACHET J AND LLOUET Cancer de la quatrième portion du duodenum Bull Acad de Med Paris 97 276 1927
- PAGET J Clinical lectures on the various risks of operations Lancet 2 1 1867
- PAINE J R History of invention and development of duodenal tube Ann Int Med 8 752 1934
- PAINE, J R The hydrodynamics of the relief of distention in the gastrointestinal tract by suction applied to intubing catheters Arch Surg 33 995 1936
- PAINE, J R AND WANGENSTEEN O H Necessity for constant suction to intubing nasal tubes for effectual decompression or drainage of upper gastro intestinal tract with comments upon drainage of other body cavities Surg Gynec & Obst 57 601 1933
- PALMER J D Volvulus of the caecum Canad M A J 60 486 1949
- PARE A The Ilorkes of that Famous Chirurgion Ambrose Parey translated by Th Johnson T Cotes and R Young London 1634
- PARKES W R AND KARABIN J E Intestinal obstruction following technical error in performance of Baldy Webster operation Am J Surg 44 659 1939
- PARKES W H AND LASHMET F H Intussusception in an adult Am J Surg 78 537 1949
- PARSONNET E V Diagnosis and management of small bowel obstruction J M Soc New Jersey 46 270 1949
- PARSON W H AND PERKS W K The elderly patient as a surgical risk Arch Surg 58 888 1949
- PARSONS W H AND WILLIAMS W T Surgical problems in the aged Negro South Surgeon 16 1163 1950
- PASCELLO L M Low bowel obstruction—roentgen diagnosis J Oklahoma M A 44 469 1951
- PATEY D H AND ASCROFT P B X ray diagnosis of acute intestinal obstruction Brit M J 2 1197 1935
- PAUL, L W Cholecysto duodenal fistula with gallstone obstruction of the small intestine a report of 2 cases Radiology 23 363 1934
- PALL M Enteric intussusception due to invagination of Meckel's diverticulum Brit M J 1: 504 1939
- PAUL, M Volvulus of the intestine with intertwining loops Lancet 2 809 1940
- PAULSON J A Anesthesia for surgical procedures for intestinal obstruction J Amer Assoc Nurse Anesth 18 230 1950
- PAULSON W O AND GARRETT L M An unusual case of intestinal obstruction Wisconsin M J 37 1001 1938
- PAVIOT J AND LEVRAIT M Double knot of Eimhorn tube within duodenum preventing its removal through pylorus unusual accident Lyon med 151 231 1933
- PEARSE H E JR Is toxemia the cause of death in uncomplicated intestinal obstruction? Ann Surg 93 915 1931
- PEARSE H E JR Experimental chronic intestinal obstruction from blind loops Surg Gynec & Obst 59 726 1934
- PENBERTON J DE J AND SAGER W W Intestinal obstruction following Baldy Webster operation S Clin North America 9 203 1929
- PENBERTHY G C JOHNSON C G AND NOER P J The treatment of adynamic ileus by gastrointestinal intubation South Surgeon 8 416 1939
- PENBERTHY G C NOER R J AND BRANSON C D Treatment of adynamic ileus by gastrointestinal intubation in children Surg Gynec & Obst 71 211 1940
- PENDERGRASS E P Role of the roentgenologic examination in the diagnosis of intestinal obstruction New England J Med 225 637 1941
- PERLMANN J J Klinische Beiträge zur Pathologie und Chirurgischen Behandlung des Darmverschlusses Arch klin Chir 137 245 1925
- PERRIN W S AND LINDSAY E C Intussusception—a monograph based on 400 cases Brit J Surg 9 46 1921
- PERRY E C AND SHAW L E On diseases of the duodenum Guy's Hosp Rep 1 171 1893
- PETERS D C AND FCKMAN W G JR Four simultaneous primary malignant neoplasms of the rectum and colon Am J Surg 90 688 1955
- PETERS J P The structure of the blood in relation to surgical problems Ann Surg 112 490 1940
- PETERS J P Water exchange Physiol Rev, 24 491 1944
- JEFFERSON W Anatomische und Chirurgische beiträge zur Gastro enterostomie Beitr klin Chir 29 597 1900
- PETTERON W L AND BLASS L W Congenital duodenal atresia U S Armed Forces M J 2 483 1951
- PETIT L A Thesis Paris 1900
- PELTZ J L A On a very remarkable case of familial polyposis of mucous membrane of intestinal tract and

- nas pharynx accompanied by peculiar pigmentation of skin and mucous membranes. *Nederl Maanfschr v geneesk* 10: 134 1921
- PITZNER, T. J. AND BARCE, J. A. A study of the blood chemistry and symptoms of colonic obstruction. *Mod Clin North America* 15: 1559 1932
- PITZNER, H. Duodenalstenose durch Ersatzstoma. *Zentralbl Chir.* 69: 629 1942
- PITZNER, J. W. AND RAY, H. B. Postoperative paralytic ileus. *Colorado Med.* 30: 200 1938
- PHANEUF, L. L. The surgical abdominal complications of pregnancy. *West J Surg.* 49: 31 1940
- PICKHART, O. C. Blind pouch growth occurring 9 years after partial colectomy with lateral anastomosis. *Ann Surg.* 97: 116 1933
- PLANT, O. H., AND MILLER, G. H. Effects of morphine and other opium alkaloids on muscular activity of alimentary canal: action on small intestine in unanesthetized dogs and man. *J Pharmacol and Exper Therap* 27: 341 1936
- POLOV, C. A case of congenital atresia of the small intestine. *Brit J Surg* 18: 333 1930
- PONOMAREV, A. Concerning the foreign bodies left in the abdominal cavity after laparotomy. *Zentralbl Chir* 55: 2137 1928
- POOL, E. H. Suction tip for aspiration in abdominal operations. *Ann Surg.* 58: 537 1913
- POOL, E. H., NILES, W. I. AND MARTIN, K. A. Duodenal stasis: duodeno-jejunostomy. *Ann Surg* 98: 587 1933
- POOL, R. M. An unusual experience with Miller Abbott tube. *Ann Surg* 130: 267 1949
- POOL, R. M. AND DUNAWANT, W. D. Volvulus of the sigmoid colon. *Ann Surg* 133: 719 1951
- POOL, R. M. AND PORTER, C. H. Intussusception due to inverted Meckel's diverticulum. *Am J Surg* 80: 368 1950
- POPE, C. F. Anorectal complications of pregnancy. *Am J Surg* 84: 579 1952
- POPPER, H. L. Diffusion of pancreatic enzymes through the intestinal wall in ileus. *Surgery* 7: 571 1940
- PORTIS, B. AND PORTIS, M. M. Experimental high intestinal obstruction: relief by irrigation and control of alkalosis: preliminary report. *JAMA* 75: 574 1925
- POTH, F. J. Succinylsulfathiazole and phthalylsulfathiazole in surgery. *J Clin Surg* 17: 773 1945
- POTH, E. J. Intestinal antiseptics in surgery. *JAMA* 153: 1516 1953
- POTH, E. J., FLEW, S. R. AND WOLFE, F. J. Treatment of recurring intestinal obstruction by the plication procedure. *Am Surgeon* 19: 24 1953
- POTTER, F. J. AND MILLER, J. M. JR. Intestinal obstruction: the role of infection in perforation of the colon. *Texas Res Biol & Med* 7: 671 1949
- POTTER, E. J. AND McCLELL, J. M. JR. Intestinal obstruction. *Ann Surg* 131: 159 1950
- POTTER, J. B. Congenital obstruction of duodenum: correction by duodeno-jejunostomy. *Northwest Med* 39: 241 1940
- LOTTS, W. J. Congenital atresia of intestine and colon. *Surg Gynec & Obst* 85: 14 1947
- IRATT, J. D. AND FALIS, I. S. Volvulus of the cecum and ascending colon. *JAMA* 89: 1225 1927
- PRAXAGORAS, DORLAND, W. A. *The American Illustrated Medical Dictionary*. W. B. Saunders Co., Philadelphia 1942
- PRESSLEY, T. A. Fluid levels as an aid to diagnosis in acute abdominal conditions. *Texas J Med* 29: 305 1933
- PRICE, J. F., MAYO, C. W. AND STEINBERG, A. G. Familial incidence of primary carcinoma of jejunum. *JAMA* 146: 797 1951
- PRISONER, A. Intestinal obstruction. *Canad MAJ.* 23: 1 1930
- PROBST, J. G. AND SACHAR, L. A. Pyloric obstruction due to gastric diverticulum. *J Missouri M Soc.* 45: 276 1948
- PROBST, J. G. AND SENTURIA, H. R. Volvulus of the sigmoid colon. *Surg Gynec & Obst* 77: 669 1943
- PRYDE, A. Ileal intussusception in an adult. *M J Australia*, 1: 648 1927
- PURNEY, C. W. Intestinal obstruction. *Virginia M Monthly* 78: 342 1951
- QUILLIAM, T. A. Volvulus of small intestine. *Brit M J* 1: 434 1946
- QUINCKE, H. Ueber Luftschlucken. *Verhandl deutsche Cong inn med* 8: 377 1889 (Wiesb.)
- QUINN, W. F. AND GIFFORD, J. H. The syndrome of proximal jejunal loop obstruction following anterior gastric resection. *California Med* 72: 18 1950
- QUIST, H. W. Diagnosis and treatment of acute intestinal obstruction. *Minnesota Med* 19: 372 1936
- QUISTGARD, P. C. The diagnosis and operability of acute intestinal obstruction. *J Missouri M A* 35: 75 1938
- RABINOWICH, N. AND FINE, J. The effect of Aureomycin on the intestine subjected to vascular injury. *Ann Surg* 135: 344 1952
- RABWIN, M. H. AND CARTER, R. A. Acute intestinal obstruction—its diagnosis by the flat x-ray film. *Calif & West Med.* 33: 483 1930
- RACK, F. J. Obstructive perforation of the cecum. *Am J Surg* 84: 527 1952
- RAGAN, C. The effect of adrenocortico-trophic hormone on rheumatic arthritis. *Am J Med.* 7: 741 1949
- RAMBO, C. M., LASKY, L. AND IDEN, J. R. An unusual cause of acute intestinal obstruction. *Ohio M J* 35: 261 1939
- RAMSTEDT, C. Zur Operation der angeborenen Pylorusstenose. *M Klin Berlin* 8: 1702 1912
- RANDALL, H. T., HABIB, D. V., LOCKWOOD, J. S. AND WERNER, S. C. Potassium deficiency in surgical patients. *Surgery* 26: 341 1949

- RANKIN F W Resection and obstruction of the colon (obstructive resection) *Surg Gynec & Obst* 1 594 1930
- RANKIN F W The value of cecostomy as a complementary decompressive operation *Ann Surg* 110 380 1939
- RANKIN F W AND JOHNSTON C G Major operations in elderly patients *Surgery*, 5 763 1939
- RANKIN L M AND EGER S A Intestinal obstruction due to gallstone *Am J Surg* 61 445 1943
- RANSHOFF J L AND HEIMAN J D The recognition and treatment of paralytic ileus *West J Surg* 41 331 1933
- RAVDIN I S Acute and chronic intestinal obstruction *Surg Gynec & Obst* 64 285 1937
- RAVDIN I S Some recent advances in surgical therapeutics *Ann Surg* 109 321, 1939
- RAVDIN I S Hypoproteinemia and its relation to surgical problems *Ann Surg* 112 576 1940
- RAVDIN I S AND GIMBEL N Parenteral protein nutrition *VA Tech Bull* 10-71 Feb 28 1951
- RAVDIN I S ZINTEL H A AND BENDER D H Adjuncts to surgical therapy in large bowel malignancy *Ann Surg* 126 439 1947
- RAVITCH M M AND McCUNE, R M JR Reduction of intussusception by barium enema *Ann Surg* 128 904 1948
- RAVITCH M M AND MORGAN R H Reduction of intussusception by barium enema *Ann Surg* 135 596 1952
- RAW S C Stricture of small bowel following strangulated hernia *Lancet* 1 460 1943
- REDICK R A AND HARRINGTON L A Roentgenologic observations in mesenteric thrombosis *Am J Roentgenol* 52 317 1944
- REED L B Thrombosis of superior mesenteric artery *Ann Surg* 74 797 1921
- PEPES R J MORAN I T AND JONES P A Right paraduodenal hernia with roentgen diagnosis and post operative recovery *Am J Roentgenol* 59 338 1948
- REGNIER E A Congenital obstruction of the third part of the duodenum in a two year old boy Operation with recovery *Minnesota Med* 18 60 1935
- REIFFERSCHIED M Serum proteins in experimental intestinal obstruction *Langenbecks Arch u Deut Schir Chir* 274 400 1933
- REINUS F Z Diagnostic criteria in strangulating obstruction of the small intestine *Ann Surg* 133 184 1951
- REMNINGTON J W The nervous system in shock Shock and circulatory homeostasis *Transactions of the First Conference Josiah Macy Jr Foundation* p 73 New York Oct 22 1951
- RENAULT cited by TREVELYAN F *Intestinal Obstruction Aetiology and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York 1899
- REYNOLDS R P AND CANTOR M O Surgical importance of carcinoid tumors of the ileum *Am J Surg* 71 705 1946
- REYNOLDS R P AND CANTOR M O Demonstration of pseudo sphincter formation at the true gastric stoma after partial gastrectomy *Grace Hosp Bull* 26 17 1948
- RIEZAES cited by MASSENGILL, S E *A Sketch of Medicine and Pharmacy* S E Massengill Co Bristol Tenn 1940
- RIBAS Y RIBAS E Intestinal obstruction from congenital deformity of the small intestine *Am J Surg* 14 215 1931
- RICHARDSON E D Acute intestinal obstruction study of a second series of cases from Massachusetts General Hospital Boston *M & S J* 183 288 1920
- RICHARDSON F P Intestinal obstruction following Baldy Webster suspension *Surg Gynec & Obst* 31 90 1920
- RIDER H Die physiologische Dickdarmbewegung beim Menschen *Fortschr Geb Rontgenstrahlen* 18 85 1912
- RIESSER J F AND VICAS B Gallstone impacted in the duodenal cap *Radiology* 58 401 1952
- RIGLER L G Roentgen observation of benign tumor of stomach prolapsing through the pylorus *Am J Roentgenol* 20 529 1928
- RIGLER L G Roentgen diagnosis of acute abdominal conditions *Bull Univ Minn Hosp* 16 120 1944
- RIGLER L G BORKAN C N AND NOBLE, J F Gallstone obstruction pathogenesis and roentgen manifestations *JAMA* 117 1753 1941
- RIGLER L G AND ERICKSEN L B Benign tumors of stomach *Radiology* 26 6 1936
- RINGSTED A AND ANDERSON K Experiments on oxygen therapy in experimental meteorism *Acta chir scandinav* 90 5,9 1945
- RINER A S Intestinal decompression—a review of methods *J Oklahoma M A* 36 197 1943
- RISTINE F R HANSEN A S AND PRINCIPATO I P Operative recovery of multiple atresia at ileo cecal area *J M Soc New Jersey* 48 65 1951
- RIVER, L P AND GLEBLER J A Transverse megacolon associated with chronic volvulus *Ann Surg* 117 786 1945
- RIVER, I P AND REED F A Volvulus of the cecum *Ann Surg* 116 874 1942
- RIVERS A B AND THIENSEN N W Obstruction of the upper portion of the small intestine *Am J Digest Dis* 1 92 1934
- ROAN O Intussusception due to benign tumors of small intestine *Texas J Med* 27 782 1932
- ROBERTS C G The modern concept of acute intestinal obstruction *Illinois M J* 67 163 1935
- ROBERTS C M AND CRANDALL, I A JR Experimental low colonic obstruction *Arch Int Med* 50 150 1932
- ROBERTSON H R Pre-operative and post-operative care of intestinal obstruction *Canad M A J*, 56 447 1947

- ROBINSON I H Mechanical partial obstruction of the colon by a pericolic diverticulum J Oklahoma M A 29 73 1935
- ROBINSON J H Surgical treatment of intestinal obstruction in preoperative preparation J Oklahoma M A 39 61 1946
- ROBINSON J R Osmoregulation in surviving slices from the kidneys of adult rats Proc Roy Soc Series P 137 378 1950
- ROBINSON J R AND McCANCE, R S Water metabolism Ann Rev Physiol 14 115 1952
- ROCKWITZ C Die Gastroenterostomie an der Strassburger chirurgischen Klinik Deutsche Ztschr Chir., 25 502 1886
- ROGERS J D, BAGGENSTOSS A H, MCGRAVE, J E AND KENNEDY R L J Intestinal obstruction associated with fibrocystic disease of the pancreas Minnesota Med 34 1075 1951
- ROGERS K E, GOLHER J C AND WILLIAMS E R Primary carcinoma of third part duodenum. Brit J Surg 40 1 1952
- ROKITANSKY C Sur les étranglements internes des intestins Arch Gen de med 14 202 1837
- ROKITANSKY C 1 Manual of Pathological Anatomy vol 2 p 52 The Sydenham Society London 1849
- ROLLESTON J D The medical aspects of Saint Simon's "Memoirs" Proc Roy Soc Med 34 31 1941
- ROMAGOSA J AND MONVILLE L J Chronic intermittent antimesenteric occlusion of the duodenum Radiology 57 63 1951
- ROMANO S A Acute mechanical intestinal obstruction New Orleans M & S J 94 270 1941
- ROMBERG R C Intestinal obstruction due to ascariasis East African M J 30 291 1953
- ROOT J C Intussusception in adult Cleveland Clin Quart 7 74 1940
- ROOT J C Intestinal obstruction Cleveland Clin Quart 16 154 1949
- ROSS P T Intussusception of the sigmoid due to simple polypus with annular carcinoma of the descending colon Brit J Surg 33 187 1945
- ROSS T F Retroposition of the transverse colon complicated by ileo caecal volvulus a report of one case with recovery and a review of the literature M J Australia 1 225 1941
- ROSEDALE R S Jejunal diverticulosis Surg Gynec. & Obst 61 223 1935
- ROSENTHAL R The child of ancient Greece Minnesota Med 19 524 1936
- ROSS G G Mesenteric thrombosis with report of 6 cases Ann Surg., 72 121 1920
- ROSS H F The surgical treatment of acute intestinal obstruction Illinois M J 74 331 1933
- ROUTLEY F F AND MAYO C W Gallstone obstruction of the small intestine Postgrad Med 12 503 1952
- RLGF E Beitrage zur Kenntnis der Darmgase Chem. Zentralbl 7 347 1862
- RUCCIERI F Calcemia ed occlusione intestinale Poli clinico (ex chir) 42 669 1937
- RUSSELL S I Diagnosis of intestinal obstruction Texas J Med 35 75 1940
- RUSELL, I A AND GRAVELLE J. J Ileocolic intussusception following appendectomy Grace Hosp Bull 29 91 1941
- RUSSELL, T H Intestinal obstruction S Clin North America 26 382 1946
- RUTHERFORD H Intestinal obstruction in newborn strangulation through a hole in mesentery Glasgow M J 71 87 1909
- RUTHERFORD H Irreducible intussusception in the infant treated by ileo-colic anastomosis Brit J Child Dis., 6 405 1909
- RYAN A J AND BURBANK J Volvulus of the cecum Am J Roentgenol 68 399 1952
- RYAN C A Intestinal obstruction due to gallstone Canad M A J., 37 167 1937
- SACHS A E Extrinsic duodenal obstruction in the newborn Ann Surg 131 117 1950
- SACHS L AND MACHT A H Intestinal obstruction with spontaneous retroperitoneal hemorrhage Bull School Med Univ Maryland 38 99 1953
- SAELTZER D V AND RHODES G K Diastasis perforation of the normal cecum resulting from obstruction of the colon Ann Surg., 101 1257 1935
- SAFRAN V AND GREENBERG A M Congenital obstruction of the transverse colon Radiology 57 738 1951
- SAGER AND ARNOLD cited by PARKES W R AND KARABIN J F Intestinal obstruction following technical error in performance of Baldy Webster operation Am J Surg 44 659 1939
- SALTZSTEIN H C CANTOR M O AND SCHEINBERG S Acute strangulating small bowel obstruction following antecolic subtotal gastrectomy Bull Sinai Hosp., 2 11 1954
- SALTZSTEIN H C AND RAO J O Large melena caused by sponge ulcerating into lumen of ileum more than 20 years after celiotomy Ann Surg 125 249 1947
- SALVATI L AND HANSON C G Incomplete rotation of the cecum with volvulus of the midgut J Internat Coll Surgeons 15 743 1951
- SALZER F Ein Vorschlag zur Modifikation der Enteranastomose durch vollige Ausschaltung des kranken Darmtheiles Ber u d Verhandl d. deutsch. Gesellsch f Chir 20 119 1891
- SAMUEL, E The cysto-gastrocolic band radiological considerations Brit J Radiol 25 19 1952
- SANDERS C B Obstructive lesions of the gastro-intestinal tract Texas J Med 30 445 1934
- SANDERSON E R Intussusception in adults Northwest Med., 51 310 1952
- SANDIFORD E Observationes Anatomicae Pathologicae Lugduni Batavorum, Eyk and Wygh 1777 1781
- SANTO L R Intestinal obstruction paralytic ileus mesenteric thrombosis and kindred lesions J Missouri M A 48 889 1951

- SARNOFF S J AND FINE J The effect of chemotherapy on the ileum subjected to vascular injury *Ann Surg.* 121 74 1945
- SARNOFF S J AND POTH E J Intestinal obstruction 1 The protective action of succinylsulfathiazole following simple venous occlusion *Surgery* 16 927 1944
- SATINSKY V P AND KRON S D Management of recurrent intestinal obstruction *Connecticut M J* 18 933 1934
- SAVIGNAC E Personal communication
- SAWYER K C The management of acute intestinal obstruction *Northwest Med* 45 733 1946
- SAWYER K C AND MARVIN H P Intestinal obstruction in infants and children *Arch Surg* 62 1 1951
- SAWYER K C NELSON J M AND McENDAFFER D M Atresia of the duodenum in a newborn child *J Internat Coll Surgeons* 13 229 1950
- SCARFF J F Aseptic end to end suture of the intestine *Ann Surg* 83 490 1926
- SCHARF A AND ACKER E Mesenteric occlusive vascular disease Presented to staff Grace Hospital March 1953
- SCHATSKI R Roentgenologic appearance of intussuscepted tumors of colon with and without barium enema examination *Am J Roentgenol* 41 549 1939
- SCHERIN C J AND BLUESTONE L Jejunal intussusception in childhood due to tumor *Surgery* 31 609 1952
- SCHERIN C J AND KRAKAUER J S Intestinal obstruction in advanced ovarian carcinoma *Am J Surg* 85 13 1953
- SCHENBERG S R AND SALTZSTEIN H C Effect of cortisone and corticotrophin (ACTH) on intra abdominal adhesions *Arch Surg* 63 413 1951
- SCHLITZMA G Permeation in the examination and treatment of the stomach and intestine *Arch Roent Ray & Allied Phenomena* 13 144 1908
- SCHIERBECK N I Ueber Kohlensäure im Ventrikel *Scandinavian Arch Physiol* 3 437 1891
- SCHIFF C A GOLDBERG S L AND NECHES A Reformation of adhesions following surgical lysis studies on dogs *Surgery* 28 977 1950
- SCHILLING J A McCOORD A B AND CLAUSEN S W Potassium loss in experimental intestinal obstruction *Surg Gynec & Obst* 92 1 1951
- SCHLICKE C E BARGEN J A AND DIXON F C The management of intestinal obstruction *JAMA* 115 1411 1940
- SCHLOERB P R, FRIS HANSEN B J, EDLMAN I S, SOLOMON A K AND MOORE F D The measurement of total body water in human subjects by deuterium oxide dilution *J Clin Invest* 29 1296 1950
- SCHLOFFER H Ueber traumatische Darmverengerungen *Mitt Grenzgeb Med Chir* 7 1 1900
- SCHLODS E M Physicochemical changes in intestinal obstruction *M Clin North America* 32 1717 1948
- SCHILLBERGER C S Plant rubber tube provided with mandarin description of new instrument and technical use *Arch mal app digest* 28 689 1913
- SCHNEIDER F H Paresis and obstruction of jejunum secondary to hysterectomy *Am J Surg* 18 85 1937
- SCHILLER F C Anatomy physiology and surgery in duodenal stasis *Minnesota Med* 24 153 1941
- SCHUMANA E A AND BECHAM C T Intestinal obstruction as a complication of the Baldy Webster uterine suspension operation *Pennsylvania M J* 42 1037 1939
- SCHUTZ C B Acute post operative obstruction of the lower small intestine *JAMA* 102 1733 1934
- SCHWARZ G Die Erkennung der tieferen Dünndarm stenosen mittels den Roentgenverfahrens *Wien klin Wchschr* 1 336 1911
- SCIORSCI E F Post operative abdominal distention *Rev Gastroenterology* 20 245 1953
- SCOFIELD P D Gallstones in the duodenum *Ann Surg* 91 632 1930
- SCOTT W W AND JARRETT J N Congenital bowel obstruction in the newborn *West Virginia M J* 42 389 1947
- SCUDDER J ZWENIER R L AND WHIFFLE A O Acute intestinal obstruction *Ann Surg* 107 161 1938
- SCULLY F J AND STELL J S Obstruction of the small intestine by enterolith recovery following removal *Southern M J* 30 93 1937
- SEABROOK D B Chronic and recurrent intestinal obstruction *West J Surg* 57 331 1949
- SEABROOK D B AND WILSON N D Prevention and treatment of intestinal obstruction by use of Noble phatation procedure *Am J Surg* 88 186 1954
- SEELAUS H K Clinical diagnosis of acute intestinal obstruction *Pennsylvania M J* 35 17 1931
- SFCEL A SCHWEINBURG F AND FINE J Effect of Sulfathaladine and sulfamerazine on gaseous distention in the obstructed small intestine of cats *Proc Soc Exper Biol & Med* 63 17 1949
- SEIDLIN S M Congenital stenosis of duodenum *Arch Pediat* 24 813 1907
- SFLAURT F E Sodium excretion by the mammalian kidney *Physiol Rev* 34 287 1954
- SFLYE H The general adaptation syndrome and the diseases of adaptation *J Clin Endocrinol* 17 358 1946
- SFNN N *Intestinal Surgery* W T Kenner Chicago 1889
- SHARBER A L Report of a case of intussusception *JAMA*, 55 907 1910
- SHATTUCK H F AND INBODEN H M Chronic intermittent duodenal obstruction *JAMA* 98 943 1932
- SHAYER W A Intestinal gas patterns in intestinal obstruction *Manitoba M Rev* 31 431 1951
- SHAW H L K AND BALDWIN L K Congenital stenosis of the duodenum *Arch Pediat* 24 813 1907
- SHAW R S AND GREFF T H JR Massive mesenteric infarction following inferior mesenteric artery ligation in resection of the colon for carcinoma *New England J Med* 248 890 1953
- SHEA P C JR Nonspecific ulcer of the small intestine *JAMA* 146 1490 1951

- SIMPSON W M Lymphatic metastases in a case of rectal adenocarcinoma simulating a clinically benign tumor *New England J Med* 215 122 1937
- SHELLFINGER P I FERRY H M AND COLFITT T S Intestinal sterilization *Guthrie Clin Bull* 22 181 1953
- SHELLEY H J Dilatation of the colon Report of a case followed by development of an aortic aneurysm re-
lieved by dilatation of the anal sphincter *Ann Surg* 109 257 1939
- SHERRY I B Acute intestinal obstruction *Am J Surg* 7 747 1929
- SHERWIN B AND ROCKEY L F Duodenal obstruction caused by chronic pylonephritis and hydronephrosis *New York J Med* 46 414 1946
- SILVER I A B Large bowel obstruction *Gynec & Obst* 63 523 1936
- SILVER S JACOB H AND CANNON J A Intestinal obstruction resulting from biliary calculi *Arch Surg* 66 301 1953
- SIRLINE K C Duodenal obstruction due to pressure of superior mesenteric vessels *JAMA* 148 550 1954
- SILKOWSKY cited by MARTIN G H Congenital anomalies of the gastrointestinal tract in infants and children *South Surgeon* 15 163 1949
- SILTZ C B Acute postoperative obstruction of the lower small intestine *JAMA* 102 1733 1934
- SEWERTH W S Intestinal obstruction due to gallstone in jejunum *Illinois M J* 58 360 1930
- SILVER P G Intestinal obstruction *Canad M A J* 22 839 1930
- SIVON H E SENTLIER H R AND KELLER T B Volvulus of the sigmoid colon *Am J Surg* 71 550 1946
- SIVON S K AND BROWN D C The medical aspect of chronic duodenal stagnation *Southern M J* 22 990 1929
- SIMPSON J Y Edinburgh *Med J* 15 390 1838
- SIMS F A WELT L G ORLOFF J AND MEDHAM J W Asymptomatic hyponatremia in pulmonary tuberculosis *J Clin Invest* 29 1545 1950
- SINGLETON A C Chronic gastric volvulus *Radiology* 34 53 1940
- SIVERTSEN I Personal communication 1946
- SKINNER G F Intestinal decompression *Canad M A J* 46 245 1942
- SKIR I Acute sigmoido-rectal intussusception. *Rev Gastroenterology* 8 232 1941
- SKJOLD A C Bowel obstruction *Minnesota Med J* 52 1948
- SLATER A S Perforation and obstruction by enterolith complicating jejunal diverticulosis *Brit J Surg* 41 60 1953
- SMITH B A JR Fever therapy in the treatment of mechanical intestinal obstruction due to pelvic inflammation *Surgery* 7 61 1940
- SMITH B B The use of Wangenstein's suction apparatus and enterostomy in abdominal surgery *Texas J Med* 37 776 1942
- SMITH B C Miller Witt tube statistical study of 1000 cases *Ann Surg* 122 253 1945
- SMITH B C AND VAN BELFEN T T Acute ileus *Ann Surg* 117 427 1943
- SMITH D W and others A case of mechanical intestinal obstruction successfully relieved by the Miller Abbott double lumen intubation tube *Jackson Memorial Hosp Bull* 2 33 1940
- SMITH I Spontaneous rupture of proximal jejunal loop as a late complication of gastrostomy *Lancet* 1 421 1953
- SMITH G A A study of intestinal intubation using a flexible stylet with controllable tip *Surgery* 32 17 1952
- SMITH G M A statistical review of variations in the anatomic position of the cecum and the processus vermiformis of the infant *Anat Rec* 5 549 1911
- SMITH H A Needle in the alimentary canal *Brit M J* 1 81 1920
- SMITH H W *The Audrey* Oxford University Press New York 1951
- SMITH R Gastro-intestinal decompression in the treatment of acute obstructions *Ann Royal Coll Surgeons England* 2 34 1948
- SMITH R Advanced treatment in postoperative ileus *Am J Surg* 19 272 1933
- SMITH R S Plication operation in treatment of small bowel obstruction *Northwest Med* 50 765 1951
- SMITH R S Leiomyosarcoma of jejunum *Am J Surg* 84 492 1952
- SMITHIES F Acute intestinal obstruction *JAMA* 95 1899 1930
- SNODGRASS T J Acute intestinal obstruction caused by non absorbable suture material *Surgery* 6 437 1939
- SNOW J M AND CLINTON M The diagnosis of intussusception by X-ray *Am J Dis Child* 6 93 1913
- SNYDER J W Chronic intestinal intussusception in the adult *South M J* 41 586 1948
- SNYDER L H Unrecognized gallstone obstruction of the intestine case report *South M J* 31 1275 1938
- SNYDER W H JR KRAUS A R AND CHAFFIN L Intussusception in infants and children *Ann Surg* 130 200 1949
- SOMERFORD A E Duodenal obstruction due to adhesions in the newborn *Lancet* 2 1309 1937
- SORLIN M Observation d'un squirre d'une portion de jejunum chez un sujet de quarante neuf ans morte a la suite de nombreux vomissements *J Gen de Med* 130 200 1949
- SORTER H BERG M AND NECHES H Constipation in the aged attempts at therapy *J Gerontol* 4 121 1949
- SOVERI V Der Verlauf der Luft durch den Verdauungskanal des Sauglings *Acta paediat* 23 1 1939
- SPAHN R R Intestinal obstruction (partial) acute nephritis and acute splenitis *Pennsylvania M J* 34 255 1931

- SPENCER J AND THAYER L T Acute obstruction of the small bowel *Radiology* 49 611 1947
- SIFACER K Intestinal obstruction in the newborn associated with faulty development of the midgut and its mesentery *Surg Gynec & Obst* 95 568 1952
- SPEARLING L The role of the ileocaecal sphincter in cases of obstruction of the large bowel *Arch Surg* 32 22 1936
- SPRONG D H POLLOCK W F AND MACK M A The anemias of intestinal stagnation *West J Surg* 61 217 1953
- STALKER L K Various types of acute intestinal obstruction surgical management *Mayo Clin Proc Staff Meet* 15 356 1940
- STANNERS F A R The management of intestinal obstruction *Ann Roy Coll Surgeons England* 9 189 1951
- STANNERS F A R Small bowel obstruction following ante colic partial gastrectomy *Brit J Surg* 40 58 1952
- STANBRO G E The diagnosis and treatment of acute intestinal obstruction *J Oklahoma M A* 33 11 1940
- STAVELY Acute and chronic gastro mesenteric ileus with cure in chronic case by duodeno jejunosomy *Bull Johns Hopkins Hosp* 19 252 1908
- STECHER W R Roentgenologic aid in the acute abdomen with special reference to intestinal obstruction *Radiology* 26 729 1936
- STEDEN E Ileus nach vorderer Gastroenterostomie (in nere Darmerklemmung) *Brunn Beitr klin Chir* 131 486 1924
- STEELE L R AND MILBURN C L JR Congenital atresia of the small intestine *U S Armed Forces M J* 3 1731 1952
- STEIN H D AND MCNEER G Rapid method for passage of Miller Abbott tube in field hospitals *Bull U S Army M Dept* 4 486 1945
- STEIN J Incomplete intestinal obstruction due to shortened ligament of Treitz *Am J Digest Dis* 2 208 1935
- STEPHENSON H U JR Intersigmoid fossa hernia *Am Surgeon* 20 1205 1954
- STETTEN D Duodenojejunosomy for congenital intrinsic total atresia at the duodenojejunal junction *Ann Surg* 111 583 1940
- STEVENS A E Two cases of atresia of the small intestine in the newborn *Arch Dis Child* 26 166 1951
- STILLMAN I R Two cases of volvulus of the caecum *Brit M J*, 2 255 1948
- STINCHFIELD F E Experimental and clinical use of oxidized cellulose and cortisone in the prevention of excess bone and fibrous tissue formation. *J Bone & Joint Surg* 32 739 1950
- STOBIE G H Obstruction of the colon *Canad M A J* 22 650 1930
- STONE G H Acute obstruction of the small bowel *Canad M A J* 24 70 1931
- STONE F Medicine among the Iroquois *Ann M Hist*, 6 529 1934
- STONE H B AND LIROR W M Absorption in intestinal obstruction intra enteric pressure as a factor *Tr Southern S A* 37 173 1924
- STONE H B AND OWINGS J C Acute mechanical intestinal obstruction treatment and results *South M J* 30 699 1937
- STORCH C REDNER B. AND TURIN R D Small bowel obstruction in infancy and childhood *J Pediat* 35 366 1949
- STOUT G Gastro intestinal obstruction—functional and organic *Ann Surg* 94 347 1931
- STOUT G The pathological physiology of ileus as a basis for treatment *Am J Surg* 19 283 1933
- STOUT G Postoperative ileus *U S Naval M Bull* 41 56 1943
- STOWERS J E Acute obstruction of the small intestine *J Missouri M A* 28 194 1931
- STREETEN D H P Control of the balance and distribution of body water *Bull Univ Hosp* 20 161 1954
- STRENGER G Intestinal obstruction due to mesenteric hiatus *Am J Surg* 78 129 1949
- STREERLIN E Zur Röntgen diagnostik der Dünndarmstenose und des Dünndarmileus *Med Klin* 1 993 1913
- STROBEL W G Acute intestinal obstruction *Minnesota Med* 15 836 1932
- STROHL E L AND PONTIUS G V Acute intestinal obstruction from biliary calculi *Illinois M J* 75 558, 1939
- STUCKEY E S Fluid and electrolyte problems in infants and children intestinal obstruction *M J Australia* 2 205 1953
- STURGEON C T Acute intestinal obstruction *S Clin North America* 13 215 1933
- STURTEVANT M Megaduodenum and duodenal obstruction *Radiology* 33 185 1939
- SUMMERS J E Enterostomy in the treatment of acute intestinal obstruction *Surg Gynec & Obst* 32 412 1921
- SWAN R C MADISSO H AND PITTS R F Measurement of extracellular fluid volume in nephrectomized dogs *J Clin Invest* 33 1447 1954
- SWEEK W O AND FRENCH G C Treatment of acute intestinal obstruction *Southwestern Med.*, 18 224 1934
- SWEEK W O AND PATTERSON J H Intestinal obstruction *Am J Surg* 7 813 1929
- SWEET J E The cause of death in high obstruction *New York J Med* 33 1194 1933
- SWEET R H Volvulus of the caecum acute and chronic *New England J Med.*, 213 287 1935
- SWENSON O Obstructions of the gastro-intestinal tract in the newborn infant *Texas J Med* 46 673 1940
- SWENSON O AND PILL A H Resection of rectum and rectosigmoid with preservation of sphincter for benign spastic lesions producing megacolon experimental study *Surgery* 24 259 1948

- SWENSON O AND LADD W I Surgical emergencies of the alimentary tract of the newborn New England J Med 233 660 1945
- SWENSON P C AND MANGES W F Roentgen findings in functional disturbances of gastro-intestinal tract Radiology 50 365 1948
- SWENSON P C AND HIRSHARD J S Roentgenographic manifestations of intestinal obstruction Arch Surg., 25 578 1932
- SYDENHAM cited by THOMAS H O *Contributions to Surgery and Medicine* H K Lewis London 1883
- SYDERHELM R Die Pathogenese der perniciösen Anämie Ergebn. inn Med u Kinderh 21 361 1922
- TACKER B Gaseous exchange between the blood and the lumen of the stomach and intestines Am J Physiol 76 92 1926
- TANNER, N C Gastro-duodenal surgery in the aged Brit M J 1 563 1943
- TANTURI C A ANDERSON R E., AND CANEPA J F Lecithinase and hyaluronidase in experimental intestinal obstruction Surg Gynec & Obst 90 171 1950
- TAPPEINER H Vergleichende untersuchung den darm gase Ztschr f Physiol Chem 6 432, 1881-82 (Strass)
- TAYLOR, F W Fluid balance and sodium chloride J Indiana M A., 45 409 1952
- TAYLOR, J H Acute intestinal obstruction and its management J South Carolina M A 26 173 1930
- TAYLOR, N B WELD C B AND HARRISON G K. Experimental intestinal obstruction. Canad. M A J 29 227 1933
- TAYLOR W HANDLEY W S AND WILKIE, D P D Acute intestinal obstruction. Brit M J., 2 993 1925
- TEASDALE, D H Colo-colic intussusception in the adult. Brit J Surg 41 128 1953
- TEITELBAUM M D The roentgen diagnosis of acute intestinal obstruction New Orleans M & S J 95 157 1942
- TEITELBAUM M D AND ARENSON N Recurrent small intestinal intussusception in children Am J Roent genol 63 80 1950
- TELFORD D Chronic arterioesenteric duodenal ileus J Internat Coll Surgeons 15 129 1951
- THEREMIN cited by TREVES F *Intestinal Obstruction Varieties and Their Pathology Diagnosis and Treatment* Wm Wood and Co New York, 1899
- THIELIS M W *Geriatrics* Mosby Co St Louis 1919
- THIESSEN N W AND ROACH F E Combined internal and external hernia with incarceration of bowel J A M A 149 1647 1952
- THOMAS C A AND HARPER F R Acute dilatation of the stomach following left sided phrenic paralysis and thoracoplasty J Thoracic Surg 5 507 1936
- THOMAS H O *Contributions to Surgery and Medicine* H K. Lewis London 1883
- THOMAS J W AND RHOADS J E Adhesions resulting from removal of serosa from an area of bowel Failure of overewing to lower incidence in the rat and guinea pig Arch Surg 61 565 1950
- THOMPSON M Chronic obstruction of the duodenum South M J 23 487 1930
- THOMPSON S Antimonyall cupps pocula emetica or calices vomitori Proc Roy Soc. Med., 19 123 1926
- THORPE P Intestinal obstruction. J Indiana M A., 40 866 1947
- THORPE P Intestinal obstruction GP 2 41 1950
- THORPE P AND LORIMER W S JR. Retrograde intussusception J A M A 133 21 1947
- THORNDAKE A Duodenal atresia and stenosis in infancy Boston M & S J., 196 763 1927
- THORNDAKE A JR., AND FERGUSON C F Incarcerated inguinal hernia in infancy and childhood Am J Surg., 39 429 1938
- TIEDEMANN F *Von der Verengung und Schliessung der Pulsadern in Krankheiten* K. Groos Heidelberg u Leipzig 1843
- TILESTON W., AND COMFORT C W The total non protein nitrogen and the urea of the blood in health and in disease by Folin's methods Arch Int. Med., 14 620 1914
- TITTEL, K. Ueber eine angeborene Missbildung des Dickdarmes Wien. klin Wchschr., 14 903 1901
- TIXIER, L. AND CLAVEL, C The retroperitoneal syndrome and the relation between kidney and gastrointestinal reflexes Surg., Gynec & Obst 54 505 1932
- TODD cited by BEST C H., AND TAYLOR, N B *The Physiological Basis of Medical Practice* 6th ed Williams and Wilkins Co Baltimore 1950
- TORREY F The causes of failure in the operative treatment of carcinoma of the esophagus Ann. Surg., 4 385 1915
- TOULROFF A S W AND SUSMAN R M Congenital prepyloric membranous obstruction in a premature infant Surgery 8 739 1940
- TRACE, H D AND GILES R C Anastomosis about the terminal ileum Am J Surg 84 473 1952
- TRENDELENBERG, F Cancer of the rectum with ster coralles fistulas In Becker E Ueber Darmresektionen Deutsche Ztschr Chir., 39 148 1894
- TRENDELENBURG P Physiologische und Pharmakologische versuche ueber die Dünndarmparastaltik Arch exper Path u Pharmacol 81 55 1917
- TREVES F *Intestinal Obstruction Varieties and Their Pathology* p 562 Wm Wood and Co New York, 1899
- TREVOR W Intestinal obstruction due to swallowed air J A M A 154 832 1954
- TROUT H H Acute intestinal obstruction. Virginia M Monthly 64 256 1937
- TRUSZKOWSKI R AND ZWEMER, R L Corticoadrenal insufficiency and potassium metabolism Biochem. J 30 1345 1936
- TUCKER, E B Intestinal obstruction mortality West Virginia M J., 33 113 1937
- TLONIKOSKI V K. Ueber die aufsteigende Dünndarmvaginatio nach der Gastroenterostomie Duodecim 15 1 1931

- TURCK F B The improved gyromele New York M J 63 191 1896
- TUREL S J Mesenteric holes or rents as a cause of intestinal obstruction Internat J Med & Surg 45 462 1932
- TURBULL R B Management of the ileostomy Am J Surg 86 617 1953
- TURNER F P AND SLOAN L W Volvulus of the small intestine in the immediate postoperative period Surgery 30 534 1951
- TURNER G C A giant gallstone impacted in the colon and causing acute obstruction Brit J Surg 20 26 1932
- TURNER J W Acute intestinal obstruction J M A Georgia 23 89 1934
- TURNER J W AND TURNER A B An analysis of fifteen cases of intussusception J M A Georgia 39 369 1950
- TURTON J R H Acute intestinal obstruction Lancet 1 708 1931
- LIU A W GROTZINGER P J SACAS C L AND MARTIN W L Diagnostic and therapeutic problems in large bowel obstruction due to carcinoma of the colon Pennskania M J 53 1290 19 0
- ULKER M Volvulus of the intestine Acta Med Turcica 2 43 1950
- UNGAR H Familial carcinoma in adolescence Brit J Cancer 3 321 1949
- URBAN K Ein Seltener Fall von Ileus Wien klin Wchnschr 42 997 1929
- URIBURU J J Jr Oclusion intestinal A proposito de dos nuevos accidentes observados en el manejo de la intubacion El Dia Medica 14 1256 1942
- URRY T V RAGLE B H ALLEN A W AND JONES C M Beriberi secondary to shortcircuited small intestine New England M J 210 251 1934
- USAPLE W Die Kreislaufstörung bei der freien eitrigen Bauchfellentzündung und der Einfluss der Darmbewegung auf den Pfortaderkreislauf Arch klin Chir 142 423 1926
- USHER F C Use of the polyethylene catheter for jejunostomy feeding Am J Surg 82 408 1951
- URZANSKI M L A new treatment for paralytic ileus Illinois M J 70 567 1936
- VAIL A D AND SCHWARTZ E J Congenital duodenal obstruction J Missouri M A 39 12 1942
- VAN ALSTYNE G S Persisting obliterated omphaloenteric duct with Meckel's diverticulum as a cause of acute obstruction Ann Surg 92 1109 1930
- VAN AMERONGEN G J The permeability of different rubbers to gases and its relation to diffusivity and solubility Rubber Chem & Technol 20 494 1947
- VAN BELLEN F T JR Symptoms and diagnosis of acute intestinal obstruction J M Soc New Jersey 29 478 1932
- VAN BELLEN F T JR Acute ileus Comparison of toxicity of obstructed and non-obstructed intestinal contents Ann Surg 102 605 1935
- VANDEL D T Fracture of ribs complicated by ileus JAMA 87 169 1926
- VANDENBERG H AND CANTOR M O Cecal volvulus in pregnancy Unpublished data
- VAN DER REIJS AND SCHIEBERA T W Weitere Studien ueber die funktionelle Darmlage operative Ergebnisse und beobachtungen am Bauchfenster Ztschr ges exper Med 52 74 1926
- VAN DUYN J Role of abdominal distention in leukocyte exhaustion Arch Surg 44 339 1942
- VAN HELMONT Ciba Symposia vol 5 1944
- VAN MEURS D P Surgical significance of aortic dissecting aneurysms Report of three personal cases with two correct ante mortem diagnoses Brit M J 2 599 1948
- VAN RAVENSWAY A Acute intestinal obstruction by large gallstone Am J Surg 16 56 1932
- VAN RAVENSWAY A C Two cases of chronic jejunal obstruction S Clin North America 15 1447 1935
- VAN ZWALENBURG C V Strangulation resulting from the distention of hollow viscera Ann Surg 46 780 1907
- VARCO R L Nutritional preparation for substandard patients Surg Gynec & Obst 84 611 1947
- VASH G, MYERS K J AND MYERS H C Pitfalls in localizing intestinal obstruction with the scout film West Virginia M J 42 1 1946
- VAUGHN A M Acute intestinal obstruction Illinois M J 97 20 1950
- VAUGHN A M Acute intestinal obstruction Chicago M Soc Bull 53 835 1951
- VAUGHN A M Epochs in the therapy of acute intestinal obstruction J Nat M A 43 93 1951
- VAUGHN A M AND NARFSTE, E M Diverticulitis of cecum Arch Surg 65 763 1952
- VELLACOTT H F Intussusception in a baby treated by resection recovery Brit M J 1 72 1942
- VELLUDA C C On a case of absence of cecum appendix ileocaecal valve and muscle tennae of ascending colon Clujul Medical 17 450 1936
- VERNEY E B Croonian lecture antidiuretic hormone and factors which determine its release Proc Roy Soc 135 25 1947
- VERNEY E B The control of water excretion Dunham Lectures Harvard University 1951
- VERON M A Medicine ancient Egypt Historical Bull Calgary Alberta 18 49 1953
- VIGAL F Quelques cas de chirurgie pancreatique Assoc Franc de Chir 18 739 1905
- VINGOFF I J Acute intestinal obstruction Ann Surg 55 801 1932
- VINGOFF I J Acute intestinal obstruction due to gall stone Am J Surg., 19 458 1933
- VIER H J Intestinal obstruction biliary calculus secondary operation recovery New York J Med 43 1233 1943

- VINA M. Retrograde intussusception of the efferent jejunal loop after gastrectomy *Arch chir uerl* 2 377 1950
- VINSON P. Cicatricial benign stricture of esophagus following vomiting and intubation *West Virginia M J* 37 349 1941
- VIRCHOW cited by BOYCE T F AND McLETRIDGE L M. Acute intestinal obstruction *South Surgeon* 6 109 1937
- VON HACKER V. cited by KFFEN W W. *An American Text Book of Surgery* W B Saunders Co. Philadelphi 1899
- VON HACKER V. Zur Casuistik und Statistik der Magenresektionen und Gastroenterostomien *Arch Klin Chir* 32 616 1885
- VON KILLICZ PARECKI. Heus and pregnancy *Munchen Med Wchnschr* 73 1352 1976
- WARD H. Intussusception of the stomach and duodenum due to a gastric polypus *Surg Gynec & Obst* 17 184 1913
- WAGNER C L, RIFFOLT F M AND SPEIR E B. Intestinal obstruction *West J Surg* 56 537 1948
- WAHRFEN H. Intestinal distension in peritonitis *Acta chir scandinav* 99 489 1950
- WAKEFIELD E G AND MAYO C W. Intestinal obstruction produced by mesenteric bands *Arch Surg* 33 47 1936
- WAKEFIELD E G, VICKERS P M AND WALTERS W. Intestinal obstruction caused by gallstones *Surgery* 5 670 1939
- WALKER, H AND KAY W. Clinico pathological reports intestinal obstruction *Virginia M Monthly* 77 247 1950
- WALLACE F T AND COLVIN F M. Operative treatment of intestinal obstruction *J South Carolina M A* 44 306 1948
- WALSTAD P M AND CARLSON E. Phrenemphraxis for the control of hematemesis in incarcerated hiatal hernia *Am J Surg* 83 567 1952
- WALTERS W AND SNELL A M. *Diseases of the Gall bladder and Bile Ducts* p 159 W B Saunders Co Philadelphia 1940
- WALTON A J. Neuromuscular obstructions of the gastrointestinal tract *Lancet* 2 1331 1930
- WANGENSTEEN O H. Acute bowel obstruction. *Minnesota Med* 14 16 1931
- WANGENSTEEN O H. The diagnosis and treatment of acute intestinal obstruction *Northwest Med* 30 389 1931
- WANGENSTEEN O H. The early diagnosis of acute intestinal obstruction *Surg Gynec & Obst* 59 785 1931
- WANGENSTEEN O H. Elaboration of criteria upon which early diagnosis of acute intestinal obstruction may be made with special consideration of value of X ray evidence *Radiology* 17 44 1931
- WANGENSTEEN O H. The early diagnosis of acute intestinal obstruction with comments on pathology and treatment *West J Surg* 40 1 1932
- WANGENSTEEN O H. Therapeutic considerations in the management of acute intestinal obstruction *Arch Surg* 26 933 1933
- WANGENSTEEN O H. Intestinal obstruction the present status of the problem *Surgery* 1 959 1937
- WANGENSTEEN O H. Rationalizing treatment in acute intestinal obstruction *Surg Gynec & Obst* 64 273 1937
- WANGENSTEEN O H. New operative techniques in the management of bowel obstruction. *Surg Gynec & Obst* 75 675 1942
- WANGENSTEEN O H. Clinical aspects of the bowel obstruction problem *Canad M A J* 54 234 1945
- WANGENSTEEN O H. *Intestinal Obstruction* Charles C Thomas Co Springfield Ill 1947
- WANGENSTEEN O H. Problems of acute intestinal obstruction *Rev Gastroenterology* 17 756 1950
- WANGENSTEEN O H AND GOEHL R O. Evaluation of the expulsion of enemas as a criterion of intestinal obstruction *Arch Int Med* 46 669 1930
- WANGENSTEEN O H AND LEVEN N L. Correlation of function with cause of death following experimental intestinal obstruction at varying levels *Arch Surg* 22 658 1931
- WANGENSTEEN O H AND LYNCH F W. Evaluation of X ray evidence as criteria of intestinal obstruction *Proc Soc Exper Biol & Med* 27 674 1930
- WANGENSTEEN O H AND PAINE J R. Treatment of acute intestinal obstruction by suction with the duodenal tube *J A M A* 101 1532 1933
- WANGENSTEEN O H AND REA C E. Distention factor in simple intestinal obstruction experimental study with exclusion of swallowed air by esophagostomy *Surgery* 5 377 1939
- WANGENSTEEN O H, REA C E, SMITH B A JR AND SCHWYZER H C. Experiences with employment of suction in the treatment of acute intestinal obstruction *Surg Gynec & Obst* 68 851 1939
- WANSBROUGH R M AND CRAW I W. Intussusception *Canad M A J* 67 307 1952
- WARD R. Appendicitis with complications reduction in mortality due to use of continuous gastrointestinal decompression *West J Surg* 48 49 1940
- WARNER, G AND SWAN H. Gallstone obstruction of small bowel occurring in the absence of a gallbladder *Surgery* 30 865 1951
- WARRFEN A S AND McQUOWN A L. Dissecting aneurysm—a presentation of ten case reports and a correlation of clinical and pathological findings *Am J M Sc* 215 209 1948
- WARREN J V AND STEAD E A JR. Fluid dynamics in chronic congestive heart failure *Arch Int Med* 73 138 1944

- WARREN K. W. AND CATTELL R. R. Stenosis of the intestine after strangulated hernia *Am J Surg* 75 729 1948
- WARTREN H. J. Acute small bowel obstruction—a study of 100 cases *Virginia M Monthly* 63 99 1936
- WASCH M. G. AND MARCK A. The radiographic appearance of the gastrointestinal tract during the first day of life *J Pediat* 32 479 1948
- WATERWORTH S. J. Intestinal obstruction *Am J Surg* 16 317 1932
- WATKINS D. H. AND MANN F. C. Motor responses of spatially transposed intestinal loops *Surgery* 25 393 1949
- WEBER H. M. The roentgenologic approach to the diagnosis of intestinal obstruction *M Clin North America* 25 1143 1941
- WEBSTER J. C. Principles and practices in the surgical treatment of retrodisplacement of the uterus *JAMA* 37 913 1901
- WEFAS C. Volvulus of a sigmoid megacolon *Ann Surg* 94 1050 1931
- WEINSTEIN M. AND ROBERTS M. Leiomyosarcoma of duodenum *Arch Surg* 66 318 1953
- WEINSTEIN, M. ROBERTS M. AND SASS E. Intestinal obstruction caused by an unusual foreign body *Gastroenterology* 14 566 1950
- WELBORN M. D. Volvulus of the cecum *J Indiana M A* 44 26 1951
- WELCH C. E. A method of constant suction applied to the Levin tube *Surgery* 6 693 1939
- WELCH C. S. Surgery in the aged *New England J Med* 238 821 1948
- WELCKER E. R. Unusual form of ileus following gastric resection *Zentralbl Chir* 75 462 1950
- WELLS C. A. AND MACPHEE I. W. The afferent loop syndrome *Lancet* 2 1189 1952
- WERSHUR L. P. Volvulus neonatorum *Am J Surg* 29 128 1935
- WEST J. P. A. AND SCHETLIN C. F. Acute mechanical obstruction of the small intestine *Am J Surg* 79 432 1950
- WESTDAHL, P. R. Hysterical abdominal distention simulating acute intestinal obstruction *California Med* 74 377 1951
- WHIPPLE, G. H. STONE, H. B. AND BERNHEIM B. M. Intestinal obstruction II. A study of a toxic substance produced by the mucosa of closed duodenal loops *J Exper Med* 17 307 1913
- WHITE C. S. AND COLLINS J. L. Congenital duodenal obstruction *Arch Surg* 43 858 1941
- WHITE J. E. M. *Ancient Egypt* pp 104–106 Thomas & Crowell Co. New York 1953
- WHITE, P. A. Obstruction of the colon by postoperative adhesions *J Iowa M Soc* 20 74 1930
- WIRBY P. F. AND COCHRAN W. H. The roentgenogram in intestinal obstruction its value and its limitations *Texas J Med* 34 740 1939
- WIKLE, H. T. Congenital obstruction of the small intestine *Am J Surg* 51 429 1941
- WILEN C. J. AND DRAGSTEIN C. A. Action of morphine on intestine in peritonitis *Proc Soc Exper Biol & Med* 28 1056 1931
- WILKIE D. P. D. Acute intestinal obstruction *Glasgow M J* 1935
- WILKINS J. A. Mercury weighted stomach tube *JAMA* 91 395 1928
- WILLARD D. G. Intestinal obstruction review of its principles *Northwest Med* 39 322 1940
- WILLENBACHER M. Fibro myoma of the stomach with intermittent ball valve action at the pylorus *Zentralbl Chir* 55 1424 1933
- WILLIAMS C. AND WILLIAMS C. JR. Surgical aspiration of the bowel in advanced obstruction *Ann Surg* 131 846 1950
- WILLIAMS C. AND WILLIAMS C. JR. Intussusception due to familial adenoma of the small intestine *Arch Surg* 59 250 1949
- WILLIAMS E. Report of a case of fecal obstruction of the small intestine with an additional plea for the earlier recognition and treatment of intestinal obstruction *N Orleans M & S J*, 61 87 1908
- WILLIAMS M. Congenital stenosis of small intestine with retention of non penetrating foreign bodies *Ann Surg* 124 492 1946
- WILLIS B. C. Acute intestinal obstruction *Am J Surg* 8 33 1930
- WILLSON D. M. Intubation experiences in medical management of patients with obstructing lesions of small and large intestine *Mayo Clin Proc Staff Meet* 15 372 1940
- WILMS M. *Der Ileus Pathologie und Klinik des Darm verschlusses* p 640 Enke Stuttgart 1906
- WILSON C. P. Foreign bodies in abdomen after laparotomy *Gynec Tr* 9 108 1884
- WILSON H. E. DESFORGES G. DUNPHY H. G. AND CAMPBELL, A. J. A. Volvulus of the cecum emphasis on possible predisposing lesions of the left colon *Arch Surg* 68 593 1954
- WINKLER A. W. DANOWSKI T. S. AND ELKINGTON J. R. The role of colloid and of saline in the treatment of shock *J Clin Invest* 25 220 1946
- WISE, R. A. The Miller Abbott double lumen tube in intestinal obstruction *Am J Surg* 41 412 1938
- WISOFF C. P. Jejunogastric intussusception *Radiology* 61 363 1953
- WOHL, M. G. BLURS J. C. AND PFEIFFER, G. High intestinal obstruction in dogs treatment with extract of adrenal cortex *Proc Soc Exper Biol & Med* 36 549 1937
- WOLF S. AND ALMI T. P. Experimental observations on cardiospasm in man *Gastroenterology* 13 471 1949
- WOLFER J. A. BEATON I. F. AND ANDERSON P. J. Volvulus of the caecum *Surg Gynec & Obst.* 74 882 1942

- WOLFF W J AND HINDMAN R Acute appendicitis in the aged Surg Gynec & Obst 94 239 1952
- WOLFFER A Leber die von Prof Billroth ausgeführten Resektionen des karcinomatösen pylorus Wien Med Presse 27 770 1881
- WOOD E H Chemical replacement in high intestinal obstruction Canad M A J, 29 415 1933
- WOOD E H Acute intestinal obstruction—appendiceal Canad M A J 38 142 1938
- WOOL O T DEBERT I AND KAIN T Endometriosis causing intestinal obstruction JAMA 130 341 1946
- WOOD I H Intestinal obstruction following gynecological operations South M J 27 30 1934
- WOODHALL B Modified double enterostomy (Mikulicz) in radical surgical treatment of intussusception in children Arch Surg 36 969 1938
- WOODHALL J cited by THOMAS H O *Contributions to Surgery and Medicine Part I Intestinal Disease* H K Lewis London 1883
- WOODRUFF H A Some toxæmias of animals due to anaerobic organisms Brit M J 1 406 1936
- WOODVATT R T AND GRAHAM F A Alimentary respiration Ohio M J 8 407 1912
- WOOLSEY J H Congenital occlusion of the small intestine Calif & West Med 42 36 1935
- WORKMAN E W AND MILLER G G A clinical review of 241 cases of obstruction of the small bowel Canad M A J 30 141 1934
- WRIGHT P J M AND TRAFFORD P A Gall stone ileus Brit J Surg, 41 6 1953
- WYATT O S Intestinal obstruction in the newborn and the infant JAMA 146 236 1951
- WYATT O S AND CHRISTOLM T C Intussusception in infants Minnesota Med 24 587 1941
- WYATT O S AND CHRISTOLM T C Acute intussusception in infancy and early childhood Journal Lancet 67 193 1947
- YATER W M Tuberculosis of the ileocaecal valve—report of an unusual case of intestinal obstruction Virginia M Monthly 56 671 1930
- YELPO A Ueber Magenatmung beim Menschen München med Wchenschr 63 1650 1916
- YOUNG W B MEFA W J AND HERRIN R C Extrinsic and intrinsic pathways concerned with intestinal inhibition during intestinal distention Am J Physiol 124 470 1939
- YOUNG E L MORRISON H R AND WILSON W E, JR Volvulus of the cecum and ascending colon New England J Med 237 78 1947
- YOUNG F R AND MCLELLER J J Atresia of the duodenum J Iowa M Soc 28 240 1938
- YOUNG W G Jejunal polyps and intussusception associated with abnormal melanin pigmentation Surgery 34 46 1953
- ZASLOW J Early postoperative mechanical intestinal obstruction following the removal of a ruptured gangrenous appendix Am J Surg 65 246 1944
- ZECH R L Anomalous pancreas as a cause of chronic duodenal obstruction West J Surg 39 917 1931
- ZEITLIN S Intestinal obstruction Radiology 33 628 1939
- ZEITLIN S AND MAZEL M S Intestinal obstruction further experiences in the use of flat abdominal films Radiology 45 267 1945
- ZIMMERMAN L M Spastic ileus Surg Gynec & Obst 50 721 1930
- ZINNINGER M M Obstructions of the second and third portions of the duodenum Surgery 12 393 1942
- ZINTEL H A RHOADS J E AND RAVIDEN I S The use of intravenous ammonium chloride in the treatment of alkalosis Surgery 14 728 1943
- ZOEGE MANTEUFFEL W Die Achsendrehungen des Coecums Verhandl deutsche Gesellsch Chir, 27 546 1898
- ZUCKERMAN S PALMER A AND HANSON D A Effect of steroid hormones on water content of tissues J Endocrinol 6 261 1950
- ZUELZER W W AND WILSON J L Functional intestinal obstruction on congenital neurogenic basis in infancy Am J Dis Child 75 40 1948
- ZWERNER R D AND TRUSZKOWSKI R Potassium A basal factor in the syndrome of cortico adrenal insufficiency Science 83 558 1936
- ZWINGER T Mercurii crudi effectus in colica spasmodica a fecum duritis alvum contumaciter abstruente oriunda Misc Acad Nat Curios, 1687 Norimb 1707 2 decuria 6 496-521 Collect Acad d Mem Dijon 7 488-491 1766

INDEX

- Abbott 325 337 340 4/9
 Abbott technic 153
 Abdominal surgery
 mechanical obstruction after 448
 Abrams 65
 Abramson 178 247
 Acetylcholine 477
 Acls 368
 Acidosis 390
 Acker 146
 Acute obstructions
 loss of water in 391
 of colon 160
 of small bowel 91
 replacement of acute loss in 395
 significant extracellular fluid loss in 395
 Adams 4 491
 Adhesions
 avoidance of technical causes of 438
 experimental prevention of 438
 prevention of 438
 prevention of by cortisone 439
 prevention of by drugs 439
 prevention of by hormones 439
 prevention of by intraperitoneal instillation 439
 prevention of by prostigmin 440
 prevention of by heparin 439
 prevention of by selective surgery 440
 prevention of obstruction by Noble plication 441
 Satinsky Kron method of management 443
 use of dicumarol in prevention of 439
 use of papain in prevention of 439
 Adler 483
 Aged
 cardiac status in 251
 intestinal obstruction in 250
 problems in management of obstruction in 250
 risks of surgery in 252
 Aird 458
 Air swallowing 293
 Albitsky 100
 Allen 406 427 472
 Aleman 451
 Alkalosis 390
 Allingham 202
 Altmeir 412
 Alvarez 484 494
 Amendola 70
 Amussat 4 469
 Amyloidosis 298
 An 139
 Anders 81
 Anderson, 131 178 295 469
 Anesthesia
 care of patient after 447
 care of patient before 447
 Angle 115
 Anomalies of intestinal rotation
 malrotation 227
 nonrotation 227
 obstruction caused by 228
 premature fixation 228
 reversed rotation 228
 Anson 176
 Antonie 41
 Antopol 483
 Anus
 anatomy of 35
 anomalies of 14
 embryology of 13
 malformations of in infancy 224
 obstruction of 197
 Appendicostomy 407
 Apsvritus 2
 Arenson 495
 Armitage 451
 Arnold 106 114
 Ascending colon anatomy of 33
 Ascroft 273
 Ashurst 4 411
 Atkinson, 483
 Atresia of colon 223
 diagnosis of 224
 treatment of 274
 Atresia of small bowel 222
 Ayers 299
 Baillie 155
 Balfour 84 186
 Balloons
 of Cantor tube 309
 of Harris tube 309

- Balloons—*Continued*
 of Miller Abbott tube 309
 prevention of gas distention in 332
 trapped in gastrointestinal tract 372
- Bands 35
- Banerji 483
- Banner 247
- Barber 86 204
- Barber 436
- Barbette Paul 3
- Bargen 368
- Barium enema
 radiologic diagnosis by 251
 therapeutic uses in obstruction 262
- Barium sulphate
 oral use of in obstruction 282
 dangers in use of 284
- Barney 114
- Baronofsky 223 285 431
- Barron 76 253 455
- Bartholin 111
- Bartlett 426 484
- Bassler 348
- Battersby 296 298 299 301 306
- Baudamant 63
- Beardsley 406
- Beaton 176
- Becker 430
- Bednfield 150
- Behng 253
- Bell 294
- Benedict 295 300
- Beneke 422
- Benvenuti 119
- Benson 217 236
- Berg 68
- Berger 143 182 368
- Berliner 384
- Bernheim 482
- Berry R E L 378
- Berti 68
- Best 304
- Bile effect of total loss of 41
- Bill 232
- Billing 323
- Binniger 223
- Birnbaum 368
- Bisgard 446 478
- Blackburn 392
- Blain 115 467 472 473
- Blackland 65
- Blind loop syndrome 424
- Block 186
- Blodgett 339
- Bloodgood 77
- Blow 439
- Block 77
- Podan 214
- body sodium 383
- Boener 77
- Bogart 150
- Bonell 411
- Bonetus 3
- Bonney 150 358
- Borman 115 272
- Borow 177
- Bosch 253
- Bouveret 293
- Bowel obstruction
 Aureomycin in 472
 lecithinase in 469
 streptomycin in 471
 sulfaguanidine in 471
 sulfasuxidine in 470
 sulfathaladine in 471
 Terramycin in 472
- Boyce 101 146
- Boyd 78
- Boyd R 218
- Boys 439 442
- Brambridge 143
- Braun 4 450
- Brayton 138
- Brenizer 366 422
- Breschet 65
- Briele 246
- Brocq 406
- Brown 79 86 493
- Brunn 368
- Bruusgaard 184 185
- Bryant 4 75
- Buchanan 79
- Bucher 119
- Buckstein 168 186
- Buckwalter 121
- Budd 170
- Burget 300
- Burk 110
- Burke 207
- Burnett 113 119
- Burnham 164
- Bursa 17
- Burstein 446
- Busse 117
- Buxton 324
- Cahn 4 144 469
- Calder 78 217
- Calihan 473
- Calisten 4
- Campbell 179
- Cantor 111 180 308 351 356 369 372
- Cantor decompression sound 354 418
- Carlini shock effect on gall stones 312
- Carcinoma of the jejunum 171

- Carcinoma of right colon management of obstruction of 432
- Cardiospasm 52 61
- Carp 109
- Carter 218 253
- Carucci 147
- Caruola 362 366
- Cave 261 283 294 286
- Cattell 141 368 406
- Cecal diverticulitis 189
incidence of 189
pathology in 189
prognosis in 189
treatment of 189
- Cecal volvulus
carcinoma of left colon as cause of 179
gas pattern in 174
management of 432
pregnancy in 247
prognosis of 179
reversed rotation in 433
- Cecostomy 403
adjunctive procedures 406
advantages of 406
disadvantages of 407
exteriorization type 404
extraperitoneal type 403
Hunt's type 403
indications for 404
technics for 404
tube type 404
- Cecum
anatomy of 33
perforation of 432
- Celsus 2
- Chaffee 322 333 365
- Chaffin 238 244
- Cholecystoduodenocolic ligament 75
- Chalfont 176
- Chapman 478 479
- Chassin 154
- Chesterman 269 482
- Chiari 70
- Chiladitis disease gas pattern in 276
- Chloride metabolism 384
- Chodoff 369
- Christensen 399
- Chronic obstruction of colon 208
anomalies as cause of 208
foreign bodies as cause of 208
- Chronic duodenal obstruction 205
adhesions causing 205
duodenal ulcer as cause of 205
ligament of Treitz as cause of 206
- Chronic gastric obstruction pedunculated tumors as cause of 205
- Chronic intestinal obstruction 204
aminoacids in 398
electrolyte requirements in 398
intravenous fat in 399
nutrition in 398
treatment of 398
vitamins in 399
- Chronic intussusception 186
- Chronic small bowel obstruction 131
adhesions as cause of 206
granuloma as cause of 136
infections as cause of 207
stricture of ileum as cause of 207
tuberculosis as cause of 133
tumors as cause of 207
- Clark Alonzo 482
- Clarke 72
- Clasen 83
- Clavel 490
- Clostradene 63
- Closure of perforated duodenal ulcer obstruction after 449
- Clyster 1
- Cockett 427
- Cohen 100 363
- Cohn 472
- Cokkinis 30 424 429
- Cole 123
- Coley 3
- Coller 324
- Collett 472
- Colocolostomy 409
- Colon
anatomy of 33
blood supply of 34
embryology of 11
physiology of 43
- Colonic obstruction
endometriosis treatment of 193
foreign bodies as cause of 167
gallstones as cause of 167
injury to colon as cause of 196
polyps as cause of 196
role of ileocecal valve in, 161
technic of examination for 160
- Colonic surgery
diagnosis of small bowel obstruction after 463
intestinal obstruction after 443
of blind efferent loop 463
obstruction at emerging colostomy 464
obstruction at colon anastomosis 464
obstruction at colostomy site 463
paralytic ileus after 448
pelvic peritoneal floor as cause of obstruction 463
- Colostomy
care of by nurse 502
de Pezzar type 408 409
end type 408

Colostomy—*Continued*

- pelvic type 409
- spur type 408
- Combalusier 293
- Comfort 303
- Congenital duodenal obstruction gas pattern in 277
- Congenital internal hernia 235
- Congenital pyloric stenosis 221
- Cooke 383
- Cooper 145
- Cordier 211
- Coughlan 451
- Counseller 178
- Courvoisier, 112
- Coury 217
- Cowley 115
- Crawford 218
- Crikelair 369
- Crooks 9
- Crossen 111
- Crowley 303
- Cruveilhier 72
- Cuenot 211 488
- Cunningham 25
- Cuny 488
- Cutting 482

Dalton's law 295

Davis 467 471

Death

- age as factor in 505
- delay in diagnosis as cause of 503
- distention as factor 504
- factors affecting 503
- location and extent of obstruction as cause of 503
- nutritional factors 505
- peritonitis as cause of 508
- plasma loss as cause of 507
- secondary causes of 508
- strangulation of bowel as factor 507
- toxic factor in 507

Decompression tube care of 501

Deglutition

- abnormalities in mechanism 36
- mechanism of 36

De Graaf 2

Demidova 303

Dennis 161 241 430

Descending colon anatomy of 33

Desforges 179

Deutsch 132

Devine 318 353

De Witt 117

Diaphragmatic hernia 55 107

congenital type 235

Dieffenbach 4

Diversitary ileocolostomy 407

Diverticulitis

- diagnosis of 189
- obstruction produced by 190
- of cecum 189
- of left colon 189
- of sigmoid with carcinoma 190
- of small bowel 145
- treatment of 190
- Dixon 179 247 368 401
- Doerflor 422
- Donhauser 139
- Doob 131
- Dott 13 227
- Douglass 247
- Dowd 241
- Drabkin 379
- Dragstedt 41 227 298 299 300 473
- Draper 165
- Drossner 477
- Dudley 25
- Dujardin Beaumetz 5 370
- Dunavant 141
- Dunlop 195
- Dunn 141
- Dunphy 179

Duodenum

- adhesions obstructing first limb 74 415
- anatomy of 22
- anomalies of 78
- arterioenteric vascular compression of 76 417
- atresia of 10
- benign tumors of 84
- carcinoma of 84
- duplication of 10
- embryology of 9
- foreign bodies in 82
- foreign bodies obstructing 416
- management of carcinoma of 415
- management of obstruction of 415
- neuromuscular disorders of 86
- obstruction by annular pancreas 416
- obstruction by benign tumor 415
- obstruction by carcinoma 416
- obstruction by diaphragm 416 417
- obstruction of 3rd limb 417
- obstruction by ulcer 86 415
- physiology of 40
- Duodenal atresia
 - diagnosis of 218
 - Mongolism with 218
 - treatment of 220
 - types of 217
- Duodenal diaphragm 78 205
- Duodenal fossae 27
- Duodenal ileus pancreatitis in 82
- Duodenal tubes
 - Ruck test 316

- Complications due to use of 321
- Contraindications to use of 321
- Cricoid chondritis from 323
- Einhorn 316 317
- Errors in use of 323
- esophageal complications from 322
- Gross 316
- Hemmeter 315
- Hess 318
- Hollender 317
- Jutte 316
- Kanavel 316
- Koslow 318
- knotting of 322
- Kuhn 317
- laryngeal complications of 323
- laryngeal stenosis from 323
- length used 319
- Levin 316
- Lyon 316
- mechanism operative in its use 319 321
- nose and nasopharynx complications from 322
- Palefsky 316
- perforation of piriform sinus from 323
- perforation of stomach from 323
- Rehlfuss 316
- safeguards in use of 324
- speed of descent 319
- stylets used in 319
- Türk 315
- Twiss 317
- Wangensteen 317
- Wilkins 317
- Dupuytren 4
- Duret, 3
- Dysphagia 52
 - hysterical type 52
 - lusoria 52
- Eaton 492
- Edelman 383
- Eger 84
- Einhorn 325
- Electrolyte concentration 382
- Electrolyte metabolism 381
- Electrolyte shock 388
- Eliason 323
- Elman 41 83
- Elsner 112
- Elsom 477
- Enteric anastomosis
 - Beri beri in 427
 - enteritis in 427
- Enterolithiasis 117
- Enterotomy 401
 - disadvantages of 403
 - Gamble method of 402
 - history of 4
- Mayo method of 401
- Witzel method of 402
- Epigastric artery rupture of simulating obstruction 486
- Erasistratus 1
- Ergotamine 477
- Ernsberger 230
- Ernst 217
- Felsing 368 372
- Esophageal
 - atresia 216
 - diagnosis of obstruction of 216
 - varices 322
- Esophagus 48
 - actinomycotic 48
 - caused by chemicals 49
 - diphtheritic 49
 - due to typhoid fever 49
 - tuberculous 48
- Esophagus
 - anatomy of 20
 - anomalies of 7
 - benign tumors of 56
 - carcinoma of 56
 - chronic granulomas of 50
 - congenital atresia of 51
 - cysts of 56
 - effect of lesions on 37
 - embryology of 7
 - foreign bodies in 58
 - inflammatory obstructions of 59
 - intubation in management of obstructions of 60
 - obstruction of 46
 - obstruction of diagnostic procedures 47
 - perforation of 51
 - psychomatic aspects 37
 - pulsion diverticulum of 53
 - role of diaphragmatic activity on, 37
 - strictures of 50
 - symptoms of obstruction 46
 - syphilitic ulcers of 50
 - traction diverticulum of 55
 - traumatic obstruction of 59
 - ulcers of 249
- Essen Moeller 249
- Estes 427
- Eudel 406
- Evans 95
- Evans W A 224
- Extracellular fluid depletion
 - effect on body 388
 - hidden losses 394
- Faber 302
- Fabricius Hildanus 110
- Fagge 174
- Failla 117
- Fair 111

- Fallis 240 247
 Farber 223
 Felmus 100 144
 Ferguson 234
 Fetal peritonitis, 230
 Feuth 247
 Figi 323
 Figiel 175 177 275
 Filho 297 300 400
 Fine 296 298 301 306 407 472 507
 Finley 353
 Firor 299 469
 Fisher 82
 Fitzwilliams 186
 Fleming 468
 Flemming 186
 Flint 422
 Floyd 237
 Foerster 126
 Folley 368 479
 Foreign bodies left in abdomen 110
 Forster 478
 Fox 218
 Franco 322
 Frank 493
 Fredet 222
 French 478
 Frick's law 306
 Friedman 195
 Fries 295
 Frimann Dahl 206 283 301
 Fritz suction 349
 Fuchs 301
 Functional intestinal obstruction
 methonium compounds a cause of, 151
 small bowel obstruction due to 150
 Furr 469
 Furth 472
 Gage, 86 401 482
 Gale 114
 Gallison 150
 Gallstone ileus 111
 intestinal gas pattern in 272
 Gamble H A 353
 Gamble 41 470
 Gardner 150
 Gas
 interchange between blood and bowel wall 294
 shadows within bowel 261
 Gaster 467 471
 Gastrectomy subtotal
 gallstone ileus after 462
 intussusception after 461
 obstruction by adhesions after 456
 obstruction by herniation of loops after 457
 obstruction of distal loop by adhesions 455
 obstruction of proximal loop after 458
 obstruction at stoma 453
 paralysis of distal jejunal loop after 454
 proximal loop volvulus after 460
 small bowel obstruction after 453
 vicious circle obstruction after 461
 volvulus after 456
 Gastric piles 66 205
 Gastric reservoir physiology of 38
 Gastric surgery
 electrolytes in 64
 intestinal obstruction after 448
 obstruction after 449
 paralytic ileus after 448
 Gastroduodenal tube
 electrolytes in use of 320
 indications for use of 320
 use of 315
 water in use of 320
 Gastroenterostomy
 closure of stoma after 451
 herniation of jejunal loops after 452
 intussusception after 452
 obstruction by axial rotation of jejunum 457
 obstruction through mesocolon 451
 prolapse of jejunum through stoma of 451
 small bowel obstruction after 449
 stomal obstruction 453
 Gastrointestinal tract
 anatomy of 18
 embryology of 7
 physiology normal of 36
 transit time of food through 44
 Gastrojejunostomy axial rotation of jejunum after 453
 Gastrostomy Janeway type 413
 Gatch 296 298 299 301, 306
 Gatellier 175
 Gaudina 381
 Gendel 298 301 507
 Gerbode 77
 Gerwig 180 197
 Gibson 5 65 90
 Gifford 459
 Giles 424 476
 Gilmore 111
 Glass 178 247
 Glenard 77
 Glick 483
 Goehl 94
 Goeller 222
 Golden 477 495
 Gollier 86
 Golub 284
 Gomco therapeutic pump 350
 Goodall 197
 Gordinier, 145
 Gordon 368
 Garelik 302

- Gould 9
 Graham 793 455
 Cranet 203
 Cravell 178 141
 Graves 482
 Gray 79
 Green 128, 147
 Gregory 76
 Greiffenlogen 182
 Crivole 5
 Gross 178 236 416 430
 Groth 183
 Gruber 148
 Grulle 230
 Guller 181
 Guibal 211 488

 Haggard 237
 Haight 7 216
 Haller 238
 Halstead 137
 Hamaker 192
 Hand, 111
 Handley 195
 Harding 467 473
 Hardy 379
 Harkins 115 137 231 473
 Harper 467 472
 Harrington 236 282
 Harris 74 141 166 368
 Hart 150
 Hartmann 85 155
 Hartwell 470
 Harvey 74 116
 Harvey S C 176
 Havens 109
 Hawthorne 469
 Hayden 401
 Hayes 399
 Heidenham, 411
 Hellens 450
 Henderson 84 468
 Henderson Hesselbach equation 390
 Henry's law 295 306
 Hernia in infancy
 incarceration of 233
 strangulation of 234
 Hernia obstructed large management of 426
 Herrin 43 296 300
 Herodotus 1
 Heschl 162
 Hiatus hernia 55
 obstruction of stomach in 414
 Hill 95
 Hilton 384
 Himshaw 472
 Hinsch 366 375
 Hippocrates 2

 Hipsley 231
 Hiratzka 369
 Hirschsprung 239 282
 Hirschsprung's disease 231
 diagnosis of 232
 pathology of 232
 Hoag 478
 Hoffman 369 478
 Hofman 457
 Hoguet 410
 Holden 353
 Holes in Broad ligament 106
 Holinger 323
 Holm 427
 Holt 506
 Hosoi 484
 Howard 141
 Hublin 452 461 477
 Hudson W A 46
 Hulton 492
 Hunt 247
 Hunter John 19 138 237
 Hurst 61
 Husccke 35
 Hutchinson, 237
 Hydrogen sulphide effect on balloons 312
 Hysterical distention 494
 diagnosis of 494

 Iagnov 105
 Iden 110
 Iglaue 323 365
 Ileocecal fossa 35
 Ileocecal valve
 anatomy of 30
 competence or incompetence of 162
 physiology of 43
 Ileostomy syndrome 464
 Ileotransverse colostomy 409
 Ileus causes of
 acute cholecystitis as cause of 492
 acute pancreatitis as cause of 492
 amyloidosis as cause of 495
 coronary disease as cause of 490
 diabetic acidosis as cause of 495
 diaphragmatic pleurisy as cause of 492
 electrolyte deficiencies as cause of 493
 fractured ribs as cause of 491
 functional causes of 494
 heart disease as cause of 490
 lead poisoning as cause of 496
 lesions of spinal cord as cause of 491
 nutritional deficiency as cause of 492
 organic disease of mesentery as cause of 495
 persistent spasm as cause of 196
 persistent spasm of colon as cause of 495
 pneumonia as cause of 492
 pyomyelitis as cause of 496

- Ileus** causes of—*Continued*
 pyelitis as cause of 490
 retroperitoneal bleeding as cause of 211
 retroperitoneal infection as cause of 489
 seminal vesiculitis as cause of 490
 uremia as cause of 490
 ureteral colic as cause of 490
 urinary system as cause of 490
 vertebral fracture as cause of 211
 vitamin deficiency as cause of 493
- Ileus** effect of
 drugs on 482
 calcium pantothenate in 483
 eserine in 484
 hypertonic saline in 483
 pituitrin in 484
 prostigmin in 484
 stigmonene bromide in 484
 thiamin in 483
- Ileus** history of 3
- Impaction** fecal 1
- Ingelfinger** 483
- Ingested foreign bodies** 109
- Inguinal hernia** in infarct 236
- Infancy**
 intestinal gas pattern with obstruction 276
 obstruction in 214
 obstruction of duodenum in 217
 obstruction of stomach in 217
- Intersigmoid fossa hernia** 108
- Intestinal activity** stimulating drugs 476
- Intestinal content** components of 296
- Intestinal decompression tube** 324
 abscess caused by 368
 Aguar tube 327
 amount of mercury used 332
 appendicitis due to 368
 Cantor tube 327 341
 care of tube after removal 346
 chondritis due to 365
 complications due to use of 365
 contra indications to use of 364
 errors in use of 373
 esophageal hemorrhage from 365
 gas in balloons of 366
 Harris tube 327 341
 history of 32
 indications for use of 368
 intussusception due to 368 377
 Johnston tube 341
 knots in 365
 Koslow tube 341
 length to be used 356
 Martinetto's method of passage 340
 Mayer's method of passage 340
 Miller Abbott tube 325 326
 Morgenstern's method of passage 339
 Morton's method of passage 340
 Morton tube 325
 Nasal hemorrhage due to 365
 obstruction due to coiling of 368
 otitis media due to 365
 passage in children 345
 passage of air filled balloon tubes 336
 passage of mercury bearing tubes 333 341
 perforation of small bowel by 368
 preparation of Cantor tube 331
 preparation of Harris tube 331
 preparation of Miller Abbott tube 330
 preparation of patient for use of 330
 removal of tube 345
 Silvertsen's method of passage 340
 stenosis of larynx due to 365
 swallowed tubes 375
 cause of 375
 effect of 376
 technic of intubation 329
 technic of passage 340
 trapped tubes
 cause of 372
 effect of 372
 treatment of 373
 types in use 320
 ulceration of small bowel mucosa due to 368
 use in atonic or paralytic ileus 358
 use in inflammatory distention group 359
 use in left colon obstruction 363
 use in mechanical small bowel obstruction 360
 use in obstruction of colon 361
 use in right colon obstruction 361
 use of stylet in 340
 use prior to elective surgery 361
 Willson's method of passage 340
- Intestinal decompression sound**
 technic of use 355
 use in acute obstruction 354
- Intestinal distention** 293
 cause of 297
 dehydration in 305
 effect of 298
 effect on blood pressure 303
 upon circulation of bowel wall 298
 on circulatory system 301
 on erythropoiesis 303
 on hematopoietic system 302
 on leukopoiesis 302
 on motility 300
 nervous exhaustion caused by 304
 renal function 303
 general effects 301
 local effects 298
 organic disease of bowel wall as cause of 298
 psychosomatic causes 298
 reflex causes 297
 cure of gas eructa, 291

- vitamin deficiency causes 297
 vomiting caused by 304
 Intestinal gas
 components of 295
 effect upon balloons of tubes 306
 radiologic diagnosis by 263
 radiologic technique of diagnosis by 263
 role of bacteria on formation of 295
 intestinal gas patterns
 diagnosis by 264
 ileocecal valve in 265
 in nonstrangulating small bowel obstruction 266
 Intestinal lumen embryology of 14
 Intestinal motility
 effect of depressing drugs on 478
 effect of anesthetic agents on 446
 effect of ascorbic acid on 478
 effect of atropine on 478
 effect of ocin on 480
 effect of proanthine on 478
 impairment by nutritional deficiency 477
 thiamin deficiency in 477
 Intestinal obstruction
 abdominal exploration in 412
 adhesions as cause of 243
 anesthesia in 444
 antibacterial agents in management of 470
 dangers in use of antibacterial agents in 474
 death in 503
 cause of 506
 diagnosis of in pregnancy 242
 disorders simulating 496
 effect of on metabolism 384
 electrolyte changes in 393
 general anesthesia in 446
 infectious as cause of in pregnancy 245
 inhalation anesthesia in 446
 intravenous anesthesia in 446
 intussusception as cause of 244
 lithopedion as cause of 247
 local anesthesia in 444
 management of 411 412
 nursing technique in 497
 neomycin in 473
 nutrition in 394
 parenteral fluids in 378
 penicillin in 472
 potassium deficits in 390
 pregnancy with 242
 appendicitis as cause of 245
 causes of 243
 hermia as cause of 244
 incidence of 242
 management of 247
 polymyositis as cause of 245
 renal disorders as cause of 246
 risk of penicillin in 477
- role of toxins in 468
 role of streptomycin in 468
 spinal anesthesia in 445
 disadvantages of 445
 transfusions in 394
 use of sulfonamides in 470
 use of drugs in 476
 water metabolism in 378
 Intestinal toxins lysozyme 468
 Intubation 314
 effect of drugs on 478
 nursing care in 497
 Intussusception
 diagnosis of 188
 entero anastomosis in 429
 exteriorization in 430
 familial polyposis as cause of 237
 foreign bodies as cause of 141
 functional 492
 in infancy
 diagnosis of 238
 treatment of 239
 intestinal gas patterns in 271
 of colon
 management of 436
 retrograde type 186
 symptomatology of 188
 Figs 483
- Jaboulay 450
 Jackson 52 423
 Jackson's membrane 12
 Jacobson 302
 James 125
 Jarre 2/8
 Jethers 128
 Jejunostomy polyethylene tube for feeding 413
 Jensen 224
 Jenkins 227
 Jensen 469
 Johner 80
 Johns 1
 Johnson 165 204 446 478
 Johnston 305 325 338 340 375
 Johnstone 85
 Jones 165 427 478 479 492
 Joseph 64
 Jutte 318
- Kalho 142
 Kampmeier 489
 Kantor 74 477
 Kapel 116
 Kaplan 378
 Karabin 106 211 488
 Karr 375
 Kaufman 77
 Kaufman 303 375

- Keene 451
 Kehr 450
 Kelley 75
 Kellogg 77
 Kelly 139
 Kennedy 384 467 473
 Kerr 244
 Kim 304
 Kinder 487
 Kirby 177
 Kirkaldy Willis 244
 Kirklin 72
 Kirsh 110 207
 Kittle 227
 Klein 284 325
 Kleinsasser 368
 Kloiber 261
 Knight 468
 Kohn 247
 Krakauer 135
 Kraus 238
 Krieg 79
 Kuhn 237
 Kuntz 211
 Kussmaul 4

 Ladd 222 236 277 430
 Lake 319
 Landzert 104
 Lane's band 12
 Langier 4
 Lashmet 380
 Lasky 110
 Latimer 110
 Latour 293
 Laufman 96 147 421 469 473
 Laurall 185
 Lawson 41
 Lawen 354
 Leaf 389
 Lee 106 144
 Left colon
 management of obstruction of 434
 occlusive vascular disease in 191
 Leichtenstern 129 237
 Leiden, 5
 Leithauser 111 297 348 453
 Lenormont 211
 Lester 195
 Levin 368
 Levitt 381
 Levrat 322
 Lewis F C 186
 Lewis 222
 Light 468 469
 Lindemuth 360
 Linder 423
 Lintlaw 188 235

 Lipperance 4
 Lippman 318
 Litre 3 431
 Loeb 119 323
 Long 105 298
 Loretz 117
 Loss of body fluids by vomiting 385
 Louw 218
 Lowden 354
 Lundberg 143 182
 Lupton, 97
 Luquet 85
 Ljall 459
 Lynch 206

 Ma 139
 Macht 125
 Mack 302
 Mackie 477
 Mackenzie 108
 Mackenzie 465
 MacFarlane 469
 Madden 370
 Maddock 294
 Magendie 293
 Magnusson 294
 Mahon 323
 Mahoney 170 302
 Maier 72
 Maingot 181
 Mainzer 165
 Malformation of anus treatment of 226
 Maluf 392
 Mamby 389
 Manelli 455
 Manery 384
 Manoil 177
 Mann 42 110 207
 Marbury 406
 Marek 59
 Marsh 471
 Marshak 195
 Martin 467 473
 Martzloff 106 300
 Mason 82 244
 Masson 5
 Matas 348
 Matignon A 350
 Matting syndrome 207
 Mayo 129 429 451 484 488 503
 Mechanical intestinal obstruction effect of drugs
 on 485
 Meckel's 477
 Meckel 136
 Meckel's diverticulum 12
 in infancy 231
 obstruction due to 231
 Meconium ileum 229

- Meconium peritonitis 230
 Meck 43 297 300
 Melamed 59
 Mellins 268 277 351
 Mentzer 115
 Mercury
 abscesses due to 369
 appendicitis caused by 368
 fecal fistula due to 369
 granuloma caused by 370
 physical properties of 371
 role in tube head 371
 use in bowel obstruction 370
 Mersheimer 365
 Mesenteric cysts 118
 Mesenteric rents 105
 Mesenteric tumors 118
 Mesenteric vascular occlusive disease 146
 Mesenterium commune 16
 Mesentery
 anomalies of 16
 embryology of 15
 rents in 30
 Meso appendix rents in 107
 Method 96 421 467 473
 Metrakos 221
 Meyer 134 179
 Michel 368
 Miller 325 446
 Miller A J 503
 Millet 298 406
 Milman 277
 Mitchell 306
 Modlin 121
 Molino 322
 Molt 323 365
 Monks 353
 Monteiro 297 300
 Moore 147 401
 Moore F D 399
 Morgan 239
 Morlock 79
 Moroni 186 231
 Morris 168
 Morrison 373 365
 Morrissey 117
 Morton 40 218 354 472
 Motor pumps 351
 Mouat 134
 Moutier 175
 Moyer 319 381 384 389
 Movvihan 104
 Mullen 101
 Murchison 111
 Murphy 112 468
 Myenteric ganglia
 anomalies of 15
 embryology of 15
 Myenteric plexuses embryology of 15
 McCaughan 41 85 451
 McClure 470
 McCollum 128
 McCune 239
 McFetridge 101 146
 McIver 97 295 300 452
 McKittick 366
 McLaughlin 117 237 437
 McLean, 106
 MacLennan 487
 McMillan 82
 McQuarrie 303
 McQuown 488
 McSwain 141
 McWatters 143
 Narat 455
 Narsete 189
 Nasopharynx anatomy of 18
 Nelaton 4
 Nelson 211
 Neurogeni obstruction of colon 195
 Newberg 380
 Nichols 78
 Noble 115
 Noble plication procedure
 complications from 442
 indications for 441
 technic of 441
 Noer 30 236 302
 Nolan 333
 Non specific ulcer of small bowel 155
 Norgaard 185
 Norris 138
 Noskin 114
 Nuck 3
 Nummi 450
 Nyborg 237
 Nygaard 437
 Obladen 247
 Obstructed bowel
 circulatory changes in wall of 505
 abdominal pregnancy as cause of 247
 adhesions as cause of in aged 257
 Obstruction in aged
 anesthetic risks in 254
 blood volume in 253
 diabetes in 253
 diagnosis in 251
 diagnosis of adhesions as cause of 257
 diagnosis of foreign bodies as cause of 257
 diagnosis of hernia in 255
 diagnosis of tumor as cause of 256
 foreign bodies as cause of 257
 hernia as cause of 255
 management during surgery 254

- Obstruction in aged—*Continued*
 neoplasms as cause of 256
 plasma protein in 252
 postoperative treatment 259
 preoperative management 254
 pulmonary disease in 253
 treatment of adhesions as cause of 257
 treatment of foreign bodies in 258
 treatment of hernia as cause of 255
 treatment of tumors as cause of 256
 use of long tube in 257
 volvulus as cause of 258
- Obstruction of colon 157
 adhesions as cause of 163
 amebic granuloma as cause of 168
 benign tumors as cause of 168
 carcinoid tumors as cause of 169
 diagnosis of 159
 diverticulitis as cause of 173 189
 endometriosis as cause of 193
 end to end anastomosis in 431
 etiology of 163
 exteriorization procedure in 431
 fecal impaction as cause of 196
 gas pattern in 272
 granuloma as cause of 168
 infectious causes of 168
 external hernia as cause of 192
 in pregnancy 246
 intussusception as cause of 186
 malignant tumors as cause of 169
 management of 430
 obstructive resection in 431
 occlusion of blood vessels as cause of 190
 sarcoma as cause of 170
 tuberculosis as cause of 168
- Obstruction of left colon
 incidence of 158
 management of carcinoma in 434
 management of diverticulitis in 435
 management of endometriosis in 437
- Obstruction of esophagus effect on body fluids 334
- Obstruction of ileum extracellular fluid depletion
 in 387
- Obstruction in infancy
 adhesions as cause of 231 236
 anomalies of intestinal rotation 227
 congenital bands as cause of 231
 congenital causes 216
 etiology of 216
 hernia as cause of 233
 intussusception as cause of 237
 management of 215
 neurogenic causes 231
 reduplications as cause of 226
 technic of examination 214
 treatment of hernia causes in 234
- Obstruction of stomach management of 413
- Occlusive vascular disease
 intestinal gas patterns in 270
 diagnosis of 191
 prognosis of 191
 Ochsner 82 261 353 401 482 483 484
 Ochsner Albert 353
 Ogilvie 195 422
 Oliver 392
 O'Loughlin 95
 Omphalocele 235
 Omphalomesenteric duct 227
 Orgel 284
 Oropharynx anatomy of 19
 Orr 85 401
 Osler 145
- Pachet 85
 Paine 299 321 349
 Palmer 37
 Pancreas annular 80
 anomalies of 10
 embryology of 9
 Pancreatic juice effect of total loss of 41
 Paraduodenal fossa hernia 104
 Paralytic ileus intestinal gas pattern in 270
 Pare 2
 Parenteral fluid therapy 391
 Parkes 106
 Patient nursing care of after surgery 502
 Patterson 353
 Paul 143
 Paviot 322
 Pearl 203
 Pearce 427
 Pemberton 106
 Penberthy 236
 Pendergrass 278
 Perforation of cecum 162
 diagnosis of 163
 Perireal hernia 108
 Peritoneal cavity free fluid in 266
 Perlmann 182
 Perrin 188 238
 Perry 82 472
 Peterson 450
 Peutz 127
 Pfeiffer 110
 Phelps 309 372
 Pickhardt 71
 Pilliet 155
 Pillore 3 431
 Plant 446
 Plastic tubes
 Dextre tube 327
 Honor Smathers tube 329
 Koslow tube 329
 Pneumoperitoneum 265
 Pollock 302

- I vol 365
 Porcher 175
 Intrastum
 extracellular function of 384
 metal stum 383
 renal excretion of 384
 symptoms of deficiency of 391
 Poth 467 470 472 474
 Pratt 247
 Praxigoras 1 411
 Pregnancy
 carcinoma of colon in 24/
 sliding hernia in 247
 small bowel tumors in 245
 Priest 76
 Pritel 471
 Properitoneal fat line 35 266
 Prostagmin 477
 Protein effect of inadequate intake of 385
 Pryde 244
 Pyle 9
 Pyloric obstruction electrolyte loss in 393
 Pyloric sphincter physiology of 39
 Pyloric stenosis 20
 diagnosis of 221
 hypersecretion in 393
 treatment of 205 222
 Pylorospasm 40
 Pylorus
 of struction of 414
 obstruction by benign tumor 414
 obstruction of by foreign bodies 414
 syphilitic stenosis of 204
 ulcer causing obstruction of 414
 Quilliam 143
 Quincke 293
 Quinn 459
 Rabinovich 472
 Rack 162
 Radiologic intestinal intubation 351
 Ragan 440
 Ragle 477
 Rames 11/
 Rambo 110
 Pamstedt 222
 Rankin 431
 Rao 110
 Ravdin 471 493
 Ravitch 239
 Rectum
 anatomy of 34
 anomalies of 14
 carcinoma in, 199
 embryology of 13
 entameba histolytica causing obstruction of 197
 fecal impaction in 200
 foreign bodies obstructing 198
 granuloma inguinal obstructing 197
 granulomas obstructing 198
 infections causing obstruction of 197
 malformation of in infancy 224
 obstruction of 197
 obstruction by metastatic growths 200
 pelvic tumors obstructing 199
 postoperative obstructions 198
 sarcoma in 199
 ulcerative colitis obstructing 198
 X ray irradiation as cause of obstruction 200
 Redfield 295 300
 Redick 282
 Reed 149
 Renault 3
 Retroperitoneal bleeding 487
 aortic aneurysm as cause of 488
 causes of 488
 diagnosis of 489
 signs and symptoms of 489
 treatment of 489
 Retroperitoneal hernia 104
 Retroperitoneal lipoma 117
 Retroperitoneal tumors 117
 Rhazes 3/0
 Rhoads 440
 Rhodes 162
 Ribas 422
 Richardson 95 106
 Rieder 186
 Right colon
 appendicostomy in obstruction of 433
 obstruction of 432
 obstructed by occlusive vascular disease 191
 Rigler 65 112 115 268 272
 Ringsted 295
 River 181
 Roach 107
 Roan 237
 Robb 302
 Robertazzi 108
 Roberts 110
 Robinson 381
 Pockey 81
 Reckwitz 450
 Rogers 86
 Rokitansky 77 174 237
 Romberg 110
 Rose 192
 Rosenfeld 298
 Rosenmond 119
 Ross 30 79 149 470
 Rowlands 478 4/9
 Puge 296
 Ruptured epigastric artery
 diagnosis of 487
 predisposing factors 486

Ruptured epigastric artery—*Continued*

- signs of 486
- symptoms of 486
- treatment of 487
- Russell 141
- Sachs 125
- Saeltzer 162
- Sager 106
- Saltzstein 110 440
- Salzer 427
- Sampson 145
- Samuel 76 273
- Sandifort 136
- Sarnoff 467 470
- Sarris 366
- Sass 110
- Savignac 208
- Seabrook 442
- Segar 383
- Seidlin 78
- Senn N 353
- Serpico 365
- Serum sodium types of lowered concentration 396
- Scharf 146
- Schatski 140
- Schatski technic 153
- Schein 135
- Scheltema 325
- Schembera 325
- Schuerbeck 293
- Schiff 440
- Schlicke 368
- Schloerb 378
- Schlosser 164
- Schwartz 108
- Schwarz 261
- Scleroderma 298
- Scully 117
- Shaw 82 147
- Shearburn 141
- Sheehan 269 482
- Shemberg 440
- Shellenberger 472
- Shelley 488
- Shen 139
- Sherman 302
- Sherwin 81
- Shilling 472
- Shorber 237
- Shrum 77
- Shukowsky 7
- Sigmoid colon anatomy of 33
- Sigmoid fossa 34
- Sigmoidorectal intussusception 188 202
- Sigmoid volvulus
 - Bruusgaard technic of management 185
 - diagnosis of 184
 - etiology of 182
 - lead poisoning as cause of 182
 - symptomatology of 184
 - treatment of 185
- Silverstein 365
- Simon 86
- Simpson 230
- Singleton 69
- Siphonage suction 347
- Slattery 154
- Slennons 244
- Slome 468
- Small intestine
 - anatomy of 28
 - anomalies of 12
 - duplication of 15
 - embryology of 10
 - normal length of 422
 - physiology of, 42
 - tumors of 125
 - vascular patterns of 30
 - treatment of atresia of 223
- Small bowel obstruction
 - adhesions in 97
 - allergy as cause of 150
 - anomalies in development as cause of 149
 - appendical causes 120
 - benign tumors as cause of 126
 - bowel resection in 422
 - carcinoid tumors as cause of 132
 - chronic granuloma as cause of 123
 - diagnosis of 92 222
 - distention in 93
 - endometriosis as cause of 133
 - entero enterostomy in 427
 - enterostomy in 422
 - etiology of 96
 - exteriorization in 428
 - herma 101
 - Hodgkin's disease as cause of 132
 - infectious causes 120
 - internal herma with 103
 - intestinal secretions lost from 394
 - intussusception of appendix 140
 - intussusception as cause of 138
 - jejunal polyps as cause of 127
 - leiomyoma as cause of 127
 - length of small bowel removed 422
 - lymphangioma as cause of 128
 - malignant tumors as cause of 129
 - management of 417 426
 - management of non strangulating obstruction 417
 - management of paralytic ileus 422
 - management of strangulation 418
 - Meckel's diverticulum as cause of 136
 - metastatic malignant growths as cause of 134
 - nausea and vomiting in 93
 - obstipation in 94

- operative procedures for 424
- pain and spinal in 92
- peritonitis as cause of 121
- result of poliomyelitis 122
- resistative measures for strangulation 420
- retroperitoneal hemorrhage as cause of 124
- sarcoma as cause of 130
- shock in 96
- signs and symptoms of 92
- strangulation in 94
- strictures as cause of 153
- tests for strangulation in 421
- tuberculosis as cause of 122
- use of barium via long tube in 287
- use of heparin in 421
- use of oxygen in 420
- use of papaverine in 421
- use of procaine in 421
- viability tests in 421
- Small bowel resection
 - history of 4
 - end-to-end anastomosis 424
 - end-to-side anastomosis in 425
 - mechanism of recovery after 473
 - side-to-side anastomosis in 474
 - tetany after 423
- Smith B. C. 324
- Smith G. 340
- Smith G. M. 176
- Smith Homer 380
- Smith R. 352
- Smith 141
- Snell 111
- Snodgrass 99
- Snyder 238
- Sodium
 - disturbances in concentration of in extracellular fluid 389
 - effects of depletion of 387
 - metabolism of 383
 - renal excretion of 393
 - replacement fluids to be given for 393
- Sorlin 129
- Sowers 277
- Spencer 79 264
- Sperling 299 302
- Sprong 302
- Sprue 298
- Stalker 284
- Stammers 457
- Stavely 77
- Steden 452
- Stedman pump 351
- Steinberg 129
- Stell 117
- Stephenson 107
- Stigmonene bromide 477
- Stinchfield 440
- Stomach
 - abnormal function of cardiac sphincter 37
 - anatomy of 21
 - anomalies of 9
 - benign tumors of 65
 - carcinoma of 65 414
 - congenital anomalies of 63
 - chronic obstruction of 204
 - effect of infection on 62
 - electrolyte loss in obstruction of 71
 - embryology of 8
 - foreign bodies in 62
 - hypertrophic pyloric stenosis in adults 7
 - intussusception in 70
 - obstruction of 61
 - diaphragmatic hernia as cause of 70
 - neurogenic 64
 - ulcers as cause of 66
 - pathologic physiology of cardiac sphincter 38
 - physiology of 37
 - sarcoma of 65
 - stenosis at cardia of 62
 - strictures caused by acid and alkali 62
 - syphilis in 62
 - tuberculosis in 62
 - tumors of 65
 - volvulus of 67 415
- Stomach tube Cerry tube 315
- Stone 299
- Stoner 95
- Storck 353
- Stout 484
- Strangulated obstruction
 - extracellular fluid loss in 394
 - intestinal gas pattern in 268
- Strenger 105
- Stricture of small bowel
 - ammonia as cause of 154
 - X ray therapy as cause of 154
- Stylets
 - Camus 319
 - Schlumberger 319
 - Smith 340
- Suckow 300
- Suction devices 347
- Suction equipment nursing care of 501
- Sullivan 40
- Surgical intubation 352
- Surgical judgment 505
- Swan 114 462
- Sweek 353
- Sweet 142
- Swenson 232 277
- Sydenham 3
- Syderhelm 302
- Tacke 295
- Tahoferro 478

- Tannenbaum 114
 Tanti 469
 Tappener 293
 Taylor 304
 Teake 227
 Teitelbaum 495
 Telford 77
 Thaxter 264
 Theremin 4
 Thiessen 107
 Thomas 5 440
 Thorndike 234
 Thornton 300
 Thoth 1
 Tiedemann 147
 Tileton 303
 Timus 105
 Titel 232
 Tixier 490
 Torek 57
 Touroff 217
 Trace 424 426
 Transverse colon anatomy of 33
 Transverse colostomy 407
 advantages of 409
 contraindications of 410
 types of 408
 Treitz ligament anatomy of 25
 Tremaine 204
 Trendelenberg 427
 Treves 30 105 115 176 181
 Trevor 167
 Trowell 493
 Trusler 259
 Truell 467 473
 Tuomikoski 461
 Turel 105 192
 Turnbull 465
 Turner 113
 Turner G C 167

 Ulcerative colitis with stenosis 194
 Umbrathor use in bowel obstruction 286
 Ungar 85
 Urecholine 477
 Uriburu 322
 Urmy 427
 Usadel 302
 Usher 413

 Valvulae conniventes 265
 Van Amerongen 309
 Van Beuren 141 468 503
 Vandel 492
 Vandenberg 180
 Van der Reis 325
 Van Dorn 303
 Van Meurs 488

 Van Zwalenburg 299
 Vaught 189 438
 Veils 35
 Ventral mesentery embryology of 9 16
 Verney 380
 Vidal 416
 Vincent 468
 Vinson 322 365
 Virchow 147
 Volvulus 142 174
 associated with intussusception 143
 congenital causes of 144
 in infancy 229
 lead poisoning as cause of 143
 pregnancy with 244
 of cecum 174
 diagnosis of 178
 lithopedion as cause of 178
 management of 178
 of left colon 181
 of sigmoid colon
 detorsion in 436
 exteriorization in 436
 management of 436
 primary resection and anastomosis in 436
 of transverse colon 180
 treatment of 180
 Von Hacker 427 449
 Von Helmont 3 293
 Von Saal 303
 Von Wahl's syndrome 186

 Wade 70
 Wadsworth 492
 Wahren 294 296
 Walters 111
 Wangenstein 6 89 94 166 266 268 269 299 318
 323 354 365 368
 Wangenstein suction 348
 Wangenstein trochar 354
 Ward 348
 Ware 128
 Warner 114 462
 Warren 141 363 488
 Wasch 276
 Water
 amount and distribution of body water 378
 factors controlling distribution of 381
 urine in 392
 gain and loss of body water 379
 renal excretion of 380
 intoxication 389
 symptoms of 390
 Waterworth 110
 Watkins 42
 Weber 282
 Webster 106
 Weeks 182

- Wernick 110
Welch 87 400
Weltv 323
Wershub 229
Whipple 303 468
White 218
Wilkie 307
Wilkie's syndrome 77
Wilkins 318
Williams 86 117 237 354
Willson 325
Wilms 30
Wilson 110 141 179 232 442
Wolfer 106
Wolfer 449
Woolf 2 429
Woodyat 293
Woolner 178
Yeats 77
Ylppo 293 294
Youmans 300
Young 179
Zeitlin 278
Zimmerman 196
Zoege Manteuffel 175
Zuckerman 381
Zuelzer 232

